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Dicofol (Kelthane) as an Environmental Contaminant: A Review

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By Donald R. Clark, Jr.

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by

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ABSTRACT.–Dicofol, an organochlorine chemical structurally similar to DDT, is used to control mites. Kelthane, the principal commercial dicofol product, contained as much as 15% DDTr (DDT, DDD, DDE, and chloro-DDT) in 1982, in addition to dicofol and its metabolites. Because resistance can develop in mites after only 1–4 years of use, many growers limit applications to no more than one per year. In the United States, sales of dicofol products total about 3 million pounds (1.4 million kg) of active ingredient annually; by comparison, DDT sales totaled 12 million pounds (5.5 million kg) annually before the 1972 ban. More than 70% of dicofol product is applied in California, Arizona, Texas, and Florida. Florida citrus crops, the biggest consumer, and California cotton receive more than half the U.S. total.

In 1986 the Environmental Protection Agency (EPA) announced that manufacturers must immediately reduce the amount of DDTr to no more than 2.5% of dicofol products, and that those with more than 0.1% DDTr could not be sold after 1 January 1989 or used after 31 March 1989. Dicofol products contributed significantly to environmental levels of DDTr, but this contribution should now be ending. Careful studies have produced no evidence of breakdown or metabolism of dicofol to DDTr. Present concern is focused on toxic effects of dicofol itself on wildlife and on dicofol's fate in ecosystems.

Dicofol has persisted in soil at least 6 years with concentrations generally less than DDT or DDE in the same samples, presumably because DDT had also been applied. Dicofol is taken up from soil by some species of plants. Dicofol sprayed on plants has persisted more than 5 months, but dicofol was less persistent than DDT.

Dicofol products were relatively nontoxic to honey bees (*Apis mellifera*). However, in laboratory tests two species of crustaceans and fathead minnow larvae (*Pimephales promelas*) experienced significant mortality at dicofol concentrations less than levels reported in San Francisco Bay. Direct lethal oral poisoning of birds or mammals in the field is unlikely because laboratory ingestion of large amounts (≥100 ppm dietary) of dicofol products for long periods (1 month–2 years) by several species caused no deaths. Young fish were generally more sensitive (LC50 in water or survival rate comparisons) than invertebrates to dicofol products; trout species were generally more sensitive than other fish. Toxicity tests (dietary or water LC50, oral or dermal LD50) with crustaceans, insects, fish, birds, and mammals showed DDT more toxic than dicofol products in 11 of 14 instances.

Reproductive effects of dicofol products demonstrated in laboratory studies include delayed hatching and deformed larvae in fathead minnows. Mallard (*Anas platyrhynchos*) and ringed turtle-doves (*Streptopelia risoria*) on dietary dicofol product showed eggshell thinning and increased cracked or broken eggs; clutch size was also reduced in doves. Eastern screech-owls (*Otus asio*) and American kestrels (*Falco sparverius*) showed eggshell thinning without serious effects on production of young. Birds seemed reproductively less sensitive to dicofol than to DDE. Because fathead minnows accumulated dicofol to levels greater than levels that thinned eggshells and from water containing less dicofol than was reported in San Francisco Bay, fish-

eating birds may risk eggshell thinning at some localities. At different dosages, dietary Kelthane-NG (amount of DDTr not given here) given to female laboratory rats reduced their insemination rate, reduced their pregnancy rate, or prevented reproduction altogether. Because dicofol residues on plants immediately after spraying have exceeded these concentrations, some wild rodents could be at reproductive risk.

Small dicofol residues (<5 ppm wet weight) have been found in free-living fish, red-winged blackbirds (*Agelaius phoeniceus*), and eggs of several bird species; DDE was several times higher in most samples. Highest dicofol residues were in eggs of eastern screech-owls from Florida citrus areas. These dicofol residue concentrations were much less than levels accumulated by captive-dosed animals. In the laboratory, ringed turtle-doves and mallard accumulated DDE in eggs more efficiently than dicofol, and there were species differences in dicofol accumulation rates among four bird species.

Chemical analyses of eggs of fish-eating birds, collected at sites where dicofol is heavily used, could help identify populations most at risk. Subsequent studies relating nest success to level of dicofol contamination in eggs might best determine whether dicofol products have adverse reproductive effects on birds.

Dicofol became the focus of much environmental concern in 1982 when it was found that miticidal products containing dicofol also contained as much as 15% DDT and its metabolites DDD, DDE, and chloro-DDT (Figure), known collectively as DDTr (Anonymous 1982, 1984a) . This initial concern resulted in efforts to produce dicofol products without DDTr, and in concern about the possible negative environmental effects of dicofol itself.

Two-thirds of the dicofol used in the United States is sold by the Rohm and Haas Company, Philadelphia, Pennsylvania, under the brand name Kelthane. Much of the literature uses "dicofol" and "Kelthane" interchangeably. Kelthane is actually a mixture of DDTr, dicofol, and dicofol metabolites (Figure) with dicofol as the principal constituent. In this review a dicofol product is referred to as "Kelthane" if it is clear that the author used a chemical bearing this label. A product is referred to as "dicofol" if this is the only term used by the author or if it seems possible, based on description in the paper, that a chemical free of DDTr might have been used. Otherwise, a product is referred to as "dicofol product." When a specific Kelthane or dicofol product is discussed, the amount of DDTr is indicated or, if the author has not given this information, the product is identified as "Kelthane-NG" or "dicofol product-NG."

Even though procedures for chemical analysis of residues resulting from use of dicofol products have changed and are continuing to change, they have nearly always quantified dicofol and its principal metabolites, DCBP (dichlorobenzophenone), DCD (dechlorodicofol), and DCBH (dichlorobenzhydrol). (Note: Evidence summarized herein strongly indicates that DDTr compounds are not metabolites of dicofol.) In most published residue results, the analysis has produced a single number that includes all of these chemicals, because until recently most analytical procedures converted dicofol and its metabolites to DCBP before quantitation (e.g., Khorram and Knight 1977; Lyman and Anderson 1979). A few early studies separated dicofol from DCBP (Menzel et al. 1961; Brown et al. 1969; Archer and Toscano 1972; Kiigemagi and Terriere 1972; Archer

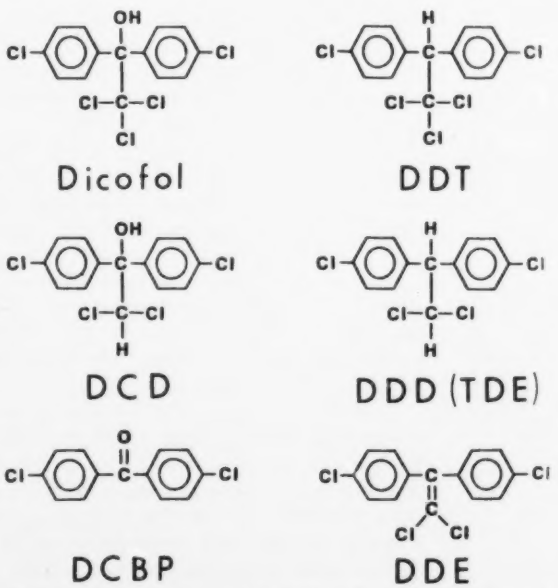

Figure. Structural formulas of dicofol, DDT, and several related compounds.

1974), but most studies that have done so are recent (Pickrell et al. 1984; Watkins et al. 1985; Brown et al. 1986; Risebrough et al. 1986; Brown and Casida 1987; Krynitsky et al. 1988; Schwarzbach et al. 1988a, 1988b; Wiemeyer et al. 1989; Beavers et al. 1989; Bennett et al. 1990; Clark et al. 1990). Some of these recent studies have also separated DCD (Brown et al. 1986; Brown and Casida 1987; Schwarzbach et al. 1988b; Beavers et al. 1989; Wiemeyer et al. 1989; Clark et al. 1990). Unless otherwise indicated, the reader should assume that the term "dicofol" in reference to specific analytical results includes dicofol plus its metabolites DCBP, DCD, and DCBH.

The term "nominal" in this text refers to the concentration of a toxicant calculated to be present in a diet on the basis of amounts mixed. The actual concentration may differ when measured by chemical analysis. When concentrations are not referred to as "nominal," they are measurements resulting from chemical analysis. Equivalent wet or dry weight residue concentrations were calculated from dry or wet weight values by division or multiplication by 3 (Heath et al. 1969; Stickel 1973). Common and chemical names of compounds are given in the Glossary.

The present report does not cite all relevant literature. Certain Russian language papers and a U.S. government report could not be obtained. I have tried to bring enough material together so that the resultant interpretations are accurate.

The purposes of the present report are to evaluate the contaminant characteristics of commercial dicofol products in light of ongoing efforts to make these products more environmentally tolerable, and render apparent the remaining questions about possible negative environmental effects of these chemicals.

What is Dicofol?

Chemistry

Dicofol is an organochlorine chemical, structurally similar to DDT (Figure), that is used to control species of mites where they are agricultural pests. Mites are arthropods of the Class Arachnida, Order Acarina. Dicofol was first registered by the U.S. Government in 1957 (U.S. Environmental Protection Agency [USEPA] 1983a). The Chemical Abstracts Registry (CAS) number for dicofol is 115-32-2. Dicofol, as produced for use in pesticides, is a waxy solid at room temperature, varying in color from dark brown to yellowish brown, stable when cool and dry, nearly insoluble in water but easily soluble in most organic solvents, and with a melting point of 58 to 78°C (USEPA 1983a). Dicofol is relatively stable to oxidation but will break down to DCBP and chloroform under alkaline conditions (March 1958).

Structurally, dicofol is DDT to which one oxygen atom has been added (Figure). To manufacture dicofol, DDT is dehydrochlorinated to DDE, which is then chlorinated to produce chloro-DDT. The chloro-DDT is then hydrolized with dilute sulfuric acid to make dicofol (Melnikov 1971).

Kelthane has been found to contain 17 constituents in addition to the *o,p'*- and *p,p'*- isomers of dicofol; these include dicofol's metabolites and the *o,p'*- and *p,p'*- isomers of DDT, DDD (Figure), DDE, and chloro-DDT (Black et al. 1971; Rothman 1980).

Efficacy

Dicofol products are effective in the control of mites (Table 1). A researcher studying cotton in California said that dicofol products also reduced populations of some insect predators of cotton pests but that possible alternative chemicals, sulfur and propargite, would have more severe effects on predator populations than dicofol (Moore 1986). Jeppson et al. (1957) reported that Kelthane-NG (FW-293) was not as effective as light–medium grade petroleum oil against citrus bud mite (*Aceria sheldoni*), and Selhime (1984) found an oil spray to be equal to Kelthane-NG in the control of the citrus rust mite (*Phyllocoptruta oleivora*).

Resistance to dicofol products is common in target species of mites (Table 2) and can develop after only 1–4 years of use (Hoyt and Harries 1961; Osakabe and Taki 1970; Saito et al. 1983). Resistance in two species (*Panonychus citri*, *Tetranychus urticae*) was caused by a single incompletely recessive gene (Overmeer and Van Zon 1973; Inoue 1980b). Normal sensitivity returned in *P. citri* when dicofol product was not used for 2–4 years (Inoue 1980b). Kornilov et al. (1980) found that sensitivity, and hence good control, of the red fruit tree mite (*T.* sp.) was maintained when Kelthane-NG was applied only once or twice each year and only when mites reached threshold densities. Fear of resistance prompted Florida citrus growers to limit their use of dicofol products to one application, or less, per year (Toner 1984).

Even though a population of mosquitofish (*Gambusia affinis*) in Mississippi had become resistant to DDT,

Table 1. *Examples of effective agricultural mite control by dicofol products.*

Location	Mite species	Year	Crop	Reference
Southern Calif.	*Panonychus citri, Brevipalpus lewisi*	1954–1956	Various citrus	Jeppson et al. (1957)
Bristol, Pa.	*Panonychus ulmi, Tetranychus telarius*	1955	Apples	Barker and Maughan (1956)
Western Tenn.	*Tetranychus* spp. (5 species) *Eotetranychus smithi*	1966	Cotton	Caldwell and Bennett (1968)
Nizhnegorskiy Rayon, Crimea, USSR	*Tetranychus* sp.	1975–1979	Apples	Kornilov et al. (1980)
Florida	*Phyllocoptruta oleivora*	1980	Oranges	Selhime (1984)
Weslaco, Tex.	*Phyllocoptruta oleivora, Eutetranychus banksi*	1983–1984	Grapefruit	French et al. (1985)

endrin, aldrin, dieldrin, toxaphene, heptachlor, lindane, and DDD from exposure to cotton agriculture, fish from this population were not cross-resistant to Kelthane-NG, which had not been used extensively on Mississippi cotton (Boyd and Ferguson 1964).

The USEPA (1984) summarized the efficacy of dicofol product: "It is efficacious against a larger number of mite species than other miticides. Dicofol does not control the citrus bud mite, but is one of only two or three pesticides effective against the board mite. Dicofol does not have temperature limitations or cause phytotoxicity problems, it is compatible with most pesticides in mixtures and is an unrestricted-use pesticide with no reentry limitations. It fits well into IPM [integrated pest management] programs because it is relatively safe to most beneficial organisms unlike the broad spectrum miticide–insecticides. Mite resistance to dicofol has been minimal over the years, whereas many of the organophosphates and others have been rendered ineffective after a few years of use."

Table 2. *Examples of resistance to dicofol products by target species of mites.*

Location	Mite species	Year	Crop	Reference
North Orondo, Wash.	*Tetranychus mcdanieli*	1959	Apples	Hoyt and Harries (1961)
Corona, Calif.	*Panonychus citri*	1959	Lemons	Jeppson et al. (1962)
Yamashiro, Japan	*Tetranychus kanzawai*	1967	Tea	Osakabe and Taki (1970)
Suwon, Kyongsan, Taegu, and Sunghwan, Korea	*Tetranychus urticae, Panonychus ulmi*	—	Fruit	Lee (1969)
Egypt	*Tetranychus arabicus, Tetranychus cucurbitacearum*	1968–1970	Cotton	Dittrich and Ghobrial (1974)
Tanushimaru and Yoshii, Japan	*Panonychus citri*	1969, 1971	Citrus	Inoue (1980a)
Central Chile	*Panonychus ulmi*	1975	Apples, pears	Campos et al. (1976)
Nizhnegorskiy Rayon, Crimea, USSR	*Tetranychus* sp.	1975	Apples	Kornilov et al. (1980)
Otago, New Zealand	*Tetranychus urticae*	1978	Strawberries	Baker (1985)
San Joaquin Valley, Calif.	*Tetranychus urticae, Tetranychus pacificus*	1982	Cotton	Dennehy and Granett (1984)

Usage

Annual sales of dicofol product in the United States are about 3 million pounds (1.4 million kg) of active ingredient (Rohm and Haas Company 1984; S. Hurt, personal communication 1990). This compares with 12 million pounds (5.5 million kg) of DDT used annually before the 1972 ban (Toner 1984). Dicofol product is used on nearly 1 million acres (0.4 million ha) in the United States each year (Toner 1984). Most dicofol product is used on citrus, cotton, and other fruit trees in California, Texas, Florida, Arizona, and upstate New York (Rohm and Haas Company 1984). More than 70% of all dicofol product is applied in California, Arizona, Texas, and Florida (Moore 1986). More than a million pounds (0.5 million kg) of dicofol product are used annually in California's San Joaquin Valley on all crops (Toner 1984). Most usage on cotton is in the San Joaquin Valley (USEPA 1983b). An estimated 100,000 pounds (45,400 kg) is used on citrus in south Texas (USEPA 1983b). The citrus industry in Florida is the biggest user; together with California cotton, these two crops receive more than half of the total 3 million pounds (Toner 1984). The Environmental Protection Agency (EPA) estimated that 60% of dicofol product is used on cotton and citrus, 10% on apples, 10% on ornamentals, commercial turf, and home gardens, and the remaining 20% on other fruits, vegetables, and field crops (Moore 1986). Seventy-three percent of dicofol product use on citrus occurs in Florida, 17% in Texas, 8% in California, and 2% in Arizona (USEPA 1984).

Between 1976 and 1986, dicofol product use on California cotton peaked in 1978; use on oranges was highest in 1976 (Table 3). Total dicofol product sales in Arizona peaked at 106,900 pounds (48,500 kg) in 1981 and declined to 31,900 pounds (14,500 kg) in 1986 (Taylor and Baker 1987).

Table 3. *Summary of recent use of dicofol products on cotton and oranges in California (California Department of Food and Agriculture 1978–1987, 1987, 1988). Actual values are × 1,000.*

Year	Cotton Pounds (kg)	Oranges Pounds (kg)
1987	194.9 (88.4)	6.0 (2.7)
1986	197.0 (89.4)	36.8 (16.7)
1985	219.1 (99.4)	15.2 (6.9)
1984	244.6 (110.9)	8.3 (3.8)
1983	190.3 (86.3)	14.5 (6.6)
1982	259.4 (117.7)	21.7 (9.8)
1981	317.3 (143.9)	18.2 (8.3)
1980	333.6 (151.3)	27.4 (12.4)
1979	343.6 (155.9)	54.0 (24.5)
1978	709.9 (322.0)	66.7 (30.3)
1977	305.1 (138.4)	74.0 (33.6)
1976	218.4 (99.1)	90.5 (41.1)

Regulatory Status

In 1982, the EPA reported that because DDTr in dicofol products was as high as 6% DDT and 9% DDE (Anonymous 1982), or 1–15% DDTr (Anonymous 1984a), and such residues might cause eggshell thinning in some wild birds, their agency had begun developing a "registration standard" for dicofol products (Anonymous 1982).

On 30 December 1983, the EPA issued "Guidance for the Reregistration of Pesticide Products Containing Dicofol as the Active Ingredient" (USEPA 1983b) and notified registrants that a "Special Review" of all dicofol products with detectable DDTr would be initiated (USEPA 1983a). At that time the EPA noted that currently registered products contained 9–15% DDTr and expressed concern even for levels <1% because of possible effects on bird reproduction (USEPA 1983a). The EPA also said that by April 1984 registrants must submit information on the composition of their products, and that by December 1986 registrants must provide test data on such subjects as environmental fate, toxicology, effects on bird reproduction and aquatic organisms, and on special testing of chloro-DDT (USEPA 1983a, 1983b).

On 21 March 1984, the EPA announced the Special Review of dicofol products and gave registrants until 7 May 1984 to rebut (Johnson 1984). If the issues could not be resolved, the EPA would propose a regulatory solution in a Position Document (PD 2/3) and, after comments from the EPA Scientific Advisory Panel, the Secretary of Agriculture, and registrants, it would give its final regulatory position. Furthermore, if the EPA determined that risks outweighed benefits, it would issue a notice of intent to cancel the registration and cancellation would occur 30 days after the notice unless the registrants requested a hearing (Johnson 1984). At this time the EPA said that dicofol products contained about 10% DDTr (Johnson 1984). In June 1984, Rohm and Haas Company reported that its products contained only 5% DDTr and that by 1985 they intended to reduce that level to 2.5% (Toner 1984).

Position Document 2/3 was issued in October 1984; the EPA said that risks outweighed benefits and it proposed to cancel all registrations for dicofol products (USEPA 1984).

On 27 March 1985, the EPA announced a meeting of the Scientific Advisory Panel to be held 16–17 April concerning the Special Review of dicofol (Moore 1985a). This announcement noted that the Rohm and Haas Company had proposed lowering DDTr in its products to 0.1%.

In August 1985, the EPA decided not to cancel the registrations of products containing dicofol because there was no good substitute, and because benefits would outweigh risks if manufacturers reduced DDTr, as they had said they could, to no more than 2.5% by 1 January 1986 and to no more than 0.1% by 1 July 1987 (Anonymous 1985a; Moore 1985b). The U.S. Fish and Wildlife Service's Office of Endangered Species (OES) rejected the EPA's decision, saying that to allow the sale of dicofol products containing more than 0.1% DDTr for 2 more years would jeopardize two endangered subspecies of the peregrine falcon (*Falco peregrinus*) and that further use should be prohibited, all existing stocks should be confiscated, and manufacture of dicofol products with more than 0.1% DDTr should cease immediately (Anonymous 1985b).

In March 1986, the OES stated that DDTr in dicofol products in the United States must be reduced to 0.1 % or less no later than 31 December 1988. An exception to this regulation provided that, in California, either (1) use and sale of all dicofol products with more than 0.1% DDTr should be stopped immediately, or (2) if sale was to continue until 31 December 1988, manufacturers should contribute to a fund supporting the Santa Cruz Predatory Bird Research Group (SCPBRG; Moore 1986). The SCPBRG would incubate peregrine eggs and place young birds in nests because peregrines in central coastal California were experiencing complete reproduc-

Table 4. *Examples of persistence of dicofol residues in surface soils.*

Location	Residue (ppm) Dicofol[a]	DCBP	Year	Years since dicofol compound was applied	Reference
Western Colorado	1.1[b]	—	1967	1	Mullins et al. (1971)
Western Colorado	1.2[b]	—	1967	1	Mullins et al. (1971)
Western Colorado	0.10[b]	—	1967	1	Mullins et al. (1971)
North-central Oregon	2.4[b]	7.2[b]	1970	3	Kiigemagi and Terriere (1972)
Southern Oregon	3.6[b]	0.86[b]	1970	4	Kiigemagi and Terriere (1972)
Southwestern Ontario, Canada	3.3[c,d]	—	1974	6	Harris and Sans (1971); Harris et al. (1977)
Washington State	0.28[d]	—	1974	3.8	Lyman and Anderson (1979)
Michigan	0.06[d,e] (ND[f]–0.19)	—	1974	4.9	Lyman and Anderson (1979)
Pennsylvania	1.0[d,e] (0.79–1.2)	—	1974	5	Lyman and Anderson (1979)
Florida	3.2[d,e] (1.9–4.1)	—	1974	2.7	Lyman and Anderson (1979)

[a] May include DCBP and DCD if separate values not given for these metabolites.
[b] Value is dry weight.
[c] Two values averaged.
[d] Value is wet weight.
[e] Three or more values averaged; range given.
[f] Not detected in chemical analysis.

tive failure due to eggshell weakness caused by DDTr (Moore 1986).

On 22 May 1986, in its "Conclusion of Special Review; notice of final determination," the EPA announced that dicofol product use could continue but that manufacturers must reduce DDTr to no more than 2.5% immediately, that all dicofol products sold after 1 January 1989 must contain no more than 0.1% DDTr, and that no dicofol product containing more than 0.1% DDTr could be used after 31 March 1989 (Moore 1986). In addition, on the basis of OES recommendation, the EPA required that dicofol manufacturers provide $325,000 to the SCPBRG over the following 5 years to pay for removal, artificial incubation, and hatching of peregrine falcon eggs in central coastal California and return of hatchlings to nests (Moore 1986). Five years was believed to be enough time for building up numbers of breeding peregrines to result in a self-sustaining population (Moore 1986). This regulation was believed sufficient to reduce environmental risks so they were clearly outweighed by economic benefits (Moore 1986). Moore also noted that DDTr in dicofol products was then between 2.5 and 5.0% (Dolan 1986), however the Rohm and Haas Company said it already was meeting the 2.5% requirement (Dolan 1986).

In September 1986, EPA ordered the Rohm and Haas Company to stop selling Kelthane and to recall any made after 29 June because it contained 2 to 3 times the 2.5% limit on DDTr set in May (Taylor and Freedman 1986). The Rohm and Haas Company responded by saying that Kelthane sold since June had been below the 2.5% DDTr limit and that the "additional material" in Kelthane was not DDTr as the EPA claimed (Taylor and Freedman 1986). However, the Rohm and Haas Company stated it had already begun the recall and that it would add another cleansing step to bring the product into compliance, although this addition would take 1–2 months (Taylor and Freedman 1986).

By October 1986, the Rohm and Haas Company had applied for registration of a purified Kelthane containing less than 2.5% DDTr (Larkin 1986). By January 1987, the Rohm and Haas Company was producing Kelthane with less than 2.5% DDTr and reaffirming that it would begin producing Kelthane with less than 0.1% DDTr in 1988 (Larkin 1987).

In 1986, the Rohm and Haas Company reported completion of studies of metabolism of C^{14}-dicofol and noted that other studies of Kelthane (<0.1% DDTr) with American kestrels (*Falco sparverius*), mallard (*Anas platyrhynchos*), and fathead minnows (*Pimephales promelas*) would be completed in 1988 (Hurt 1986). All of these studies were made to meet the EPA data request of 16 April 1986. On 12 September 1986, the EPA ordered annual monitoring of birds, fish, aquatic and terrestrial nontarget invertebrates, soil, water, and sediment for dicofol residues during the following 10 years (Larkin 1987). In 1988, the Rohm and Haas Company completed draft protocols for monitoring studies to begin in 1989 (S. S. Hurt, Rohm and Haas Co., Spring House, Pennsylvania, personal communication).

Environmental Persistence

Soils

In a test of biodegradability that used a bacterial suspension, Kelthane-NG, DDT, and methoxychlor were susceptible to degradation, whereas endrin, dieldrin, aldrin, heptachlor epoxide, lindane, and HCB (hexachlorobenzene) were not (Van Duck and Van de Voorde 1976). On this basis, persistence in soil might seem less likely for the first three compounds. However, Edwards (1985) was incorrect when he reviewed the literature on environmental dicofol residues and concluded that dicofol is not persistent in soil, because persistence in soil had been well documented since the 1970's (Table 4). The EPA had summarized, "field studies show that dicofol persists in soils for at least 4 years after application" (USEPA 1983a).

In Ontario, Canada, an apple orchard with sandy loam soil became a public park in 1968, and applications of dicofol product are presumed to have ceased (Harris and Sans 1971; Harris et al. 1977). Dicofol residues declined 33% (from 4.9 to 3.3 ppm, wet weight) between 1969 and 1974 (DDT declined 12% from 76 to 67 ppm, DDD declined 60% from 2.0 to 0.8 ppm, and DDE increased 31% from 8.4 to 11 ppm), but they had persisted 6 years after the last application and probably would have been found in subsequent years had samples been taken.

Soils at 14 of 17 agricultural sites in five States (California, Florida, Michigan, Pennsylvania, and Washington) sprayed with Kelthane-NG contained residues of dicofol when tested in 1974 (Lyman and Anderson 1979). Four sites had not received Kelthane-NG for 2.7–5 years, yet dicofol was present.

Kiigemagi and Terriere (1972) quantified dicofol and DCBP in sandy loam soil of a Hood River, Oregon, orchard 3 years after the last dicofol product spray (Table 4). The measurement for DCBP (7.2 ppm, dry weight) was large and possibly incorrect; DDT was 39, DDE 7.6, and DDD 1.6 ppm. In clay soil of a Medford,

Oregon, orchard dicofol and DCBP residues were found 4 years after spraying (Table 4); DDT was 17, DDE 2.5, and DDD 1.4 ppm. Both orchards had also been heavily treated with DDT. On the basis of their residue measurements, Kiigemagi and Terriere (1972) proposed that either dicofol and DCBP were more stable than DDT or they were metabolites or degradation products of DDT. The latter possibility gains support from Lichtenstein et al. (1971).

Lichtenstein et al. (1971) found "trace" and 0.14 pounds/acre (0.16 kg/ha) of dicofol in silt loam soils treated 15 years earlier with 10 pounds/acre (11 kg/ha) or 100 pounds/acre (112 kg/ha) of DDT, respectively. DDT residues were 1.0 pound/acre (1.1 kg/ha) from the 10-pound application and 17 pounds/acre (19 kg/ha) from the 100-pound application; DDE residues were 0.74 pounds/acre (0.83 kg/ha) and 7.2 pounds/acre (8.1 kg/ha), respectively. Finding dicofol where only DDT had been applied suggests that there could be additional records of dicofol in soil with this origin, but if this breakdown can occur, would dicofol not have been found much more often in soil? Kuhr et al. (1974) looked unsuccessfully for dicofol in soils of apple orchards that had received DDT sprays. The plots of Lichtenstein et al. (1971) received especially heavy applications and were undisturbed for 10 years; perhaps these factors resulted in the unusual finding.

Residues of 199 ppm (wet or dry weight not indicated) dicofol in sediment near Buckeye, Arizona (Kepner 1986), probably resulted from spillage or washing of tanks of applicator aircraft. It is likely that such high residues would be readily picked up by nearby aquatic food chains. The EPA reported that dicofol may accumulate in "some rotational crops" (USEPA 1983a); that is, dicofol can accumulate in crops planted in previously sprayed soils (Anonymous 1984b).

Dicofol in soil is probably not inert. A study in the Soviet Union demonstrated that Kelthane-NG inhibited two soil enzymes, invertase and catalase, to a depth of 5 cm for at least 15 days (Stepanyan et al. 1984). The ecological significance of this inhibition is unknown.

There has been no direct comparison of dicofol's persistence in soil with that of other organochlorine insecticides in which initial applications were equal. However, residues of DDT, DDE, and dieldrin in soil were clearly elevated above background residue levels 11 years after single applications of the parent compounds (Beyer and Gish 1980). Thus the known intervals of persistence for dicofol in soil are less than those for the other chemicals.

In summary, even though dicofol concentrations generally have been less than those of DDT and DDE in the same samples, dicofol has been routinely measured in soils of orchards and fields where it was applied, it has persisted for at least 6 years, when it measured 3.3 ppm (wet weight), and it is taken up by some types of plants. These factors indicate a potential for contamination of food chains.

Plants

Persistence of dicofol has been investigated in agricultural crops and plant products that are sprayed directly. Many of these studies measured how rapidly dicofol concentrations decline to legal limits on perishable fruits and vegetables, which means persistence was measured for only brief periods after spraying. Nevertheless, the results may be helpful in evaluating the possible ecological effects of dicofol.

In South Africa, Van Dyk (1975) found that whole grapefruit and oranges contained 2.3 and 11 ppm (probably wet weight) dicofol 160 days after spraying, and that oranges from other plots contained 2.5 and 2.6 ppm dicofol 134 days after spraying. Because these concentrations were for whole fruit and the author determined that all residue was in the rind, concentrations in rind itself must have been considerably higher. Van Dyk (1975) measured half-lives of as little as 4.2 days and as much as 43.5 days; he concluded that higher temperatures decreased persistence but that sunshine and ultraviolet light had no influence.

Working in California with residues in citrus rind, Gunther et al. (1957) learned that dicofol declined from 7 to 35% by 103 days postspray in samples of oranges, and from 16 to 29% by 31 days postspray in lemons. Concentrations in oranges began in the range of 5.3 to 11 ppm (wet or dry weight not indicated) and ended in the range of 3.1 to 7.8 ppm; in lemons concentrations began at 6.5 to 16 ppm and ended at 6.1 to 12 ppm. Half-lives for dicofol were estimated at 170 to 350 days in oranges and 120 to 150 days in lemons.

In the laboratory, Archer and Toscano (1972) determined that dicofol residues in apple pomace kept in darkness declined 46% from 710 to 380 ppm (dry weight), residues in apple pomace exposed to ultraviolet light (2,537Å) declined 73% from 710 to 190 ppm, and residues in apple pomace exposed to sunlight declined 48% from 710 to 370 ppm. The experimental period was 14 days for all samples. Archer and Toscano (1972) concluded that loss of residue was due more to volatilization than to photodecomposition.

Under field conditions at Masisskiy Rayon in the Soviet Union, dicofol residues were measured on tomato

fruits and leaves (Stepanyan et al. 1984). Residues in fruits declined 69% from 2.9 to 0.90 ppm (wet weight) by 5 days postspray and were not detectable at 15 days. Residues on leaves declined 92% from 94 to 7.5 ppm (wet weight) by 5 days and also were not detectable at 15 days.

In Jordan, dicofol residues on fruits of cucumbers grown under plastic covers declined to undetectable levels by 8 days postspray in three of four tests after beginning at 0.95, 1.2, and 1.3 ppm (probably wet weight); in the fourth test, residues declined 79% from 1.6 to 0.33 ppm (Nazer and Masoud 1986). Declines in dicofol residues on cucumber leaves by 8 days postspray ranged from 29 to 66% with beginning residues ranging from 48 to 400 ppm and final residues ranging from 20 to 190 ppm.

Dicofol residues on strawberries in Kentucky declined 56% from 1.8 to 0.8 ppm (probably wet weight) by 8 days postspray in one test and 62% from 0.8 to 0.3 ppm in the same interval in another test (Fahey et al. 1962). On other strawberries, DDT was more persistent, remaining at 1.4 ppm during the same 8-day interval in one test and declining only 17% from 3.0 to 2.5 ppm in another test.

In India, dicofol on tea leaves declined 82% from 13 to 2.3 ppm (probably wet weight) by 10 days postspray; the half-life was estimated at 4.1 days (Rajukkannu et al. 1981). In another Indian study, dicofol residues on two samples of tea measured 2.1 and 3.1 ppm (leaves air dried) at 14 days postspray (Singh and Agnihotri 1984).

Dicofol residues on peach leaves in Ontario, Canada, decreased 40% from 0.90 to 0.54 $\mu g/cm^2$ by 18 days postspray (Herne and Chiba 1975). The authors discovered that the mortality of European red mites (*Panonychus ulmi*) exposed to sprayed leaves fell to that of controls by day 14, even though dicofol residues had declined less than 50%. They attributed the loss of toxicity either to penetration of the dicofol into the leaf or to adsorption of dicofol to the adjuvants contained in the formulation.

In summary, the data on persistence of dicofol in or on plants show that even though declines in residues often exceed 50% in the first days postspray, in one instance substantial residues (11 ppm, probably wet weight) existed 5 months later. Loss of residues may be due primarily to volatilization, which is accelerated by higher temperatures. In a direct comparison of residues with DDT on strawberries, DDT seemed to be more persistent than dicofol. Highest levels of persistent dicofol residues were on citrus fruits. Persistence in and on plants for weeks or months allows dicofol residues to be eaten by herbivores; unless those residues are metabolized by the herbivores, they will be passed along in food chains.

Human Diet

Dicofol measured by the U.S. Food and Drug Administration in samples of adult human diet in the United States led to estimated average daily dicofol intakes of 0.005, 0.007, 0.012, and 0.006 µg/kg of body weight during fiscal years 1978, 1979, 1980, and 1981–82, corresponding values for DDTr (0.070, 0.093, 0.034, and 0.034 µg/kg per day) were 3 to 14 times greater (Gartrell et al. 1986a). The officially acceptable intake of dicofol is 25 µg/kg per day (Gartrell et al. 1986a). Amounts of dicofol found in diets of infants and toddlers were generally less than for adults (Gartrell et al. 1986b). Dicofol was reported in three categories of plant products (legumes, fruit, and sugar), but not in animal product categories of dairy, meat, fish, poultry, and fats (Gartrell et al. 1986a, 1986b). A "market basket survey" by the Natural Resources Defense Council found dicofol second only to DDTr as the most common pesticide residue in California produce (Graham 1984).

Dicofol is considered "at least possibly carcinogenic to man" (Holder 1986), but "there is only limited evidence at this time to support a conclusion that dicofol is a potential human carcinogen" (Moore 1986).

The presence of dicofol in human foods suggests that it is relatively persistent, but its absence from foods of animal origin may indicate rapid metabolism by herbivores.

Toxicity

Plants

According to the EPA, "dicofol does not have temperature limitations or cause phytotoxicity problems" (USEPA 1984). Later, it judged dicofol to have "fewer phytotoxicity problems than other miticides" (Moore 1986). Several cases of phytoxicity have been reported.

When Kelthane-NG was applied to 4- to 6-week-old cotton plants of wilt-susceptible varieties that had been innoculated with wilt (*Verticillium albo-atrum*), the time to first symptom was shortened and the severity of symptoms was increased in greenhouse conditions (Mussell and Ramftl 1972). Daley et al. (1979) demonstrated that Kelthane-NG inhibited a cell-wall enzyme in cotton of wilt-susceptible and nonsusceptible varieties

and suggested that this inhibition could benefit the fungus; they also noted that the effect could be due to an "active impurity" in the Kelthane-NG.

Thomson (1982) warned that Kelthane-NG injured eggplants and pears and that it should not be used on canaeti juniper (var. of *Juniperus virginianus*) or Chinese holly (*Ilex cornuta*).

From 1972 to 1979 at Nizhnegorskiy Rayon in the Crimea of the Soviet Union, Kelthane-NG was applied to apple orchards five or six times each growing season (Kornilov et al. 1980). By late June or early July, the trees turned dull gray, lost as much as 40% of their chlorophyll, and had reduced yield. These effects disappeared when the chemical was used only once or twice per year (Kornilov et al. 1980).

Multiple sprays of Kelthane-NG caused mild chlorosis in greenhouse apple trees but did not affect fruit growth of orchard apples in Washington State (Westwood et al. 1960). In a greenhouse in Ohio, apple trees were sprayed with dicofol product-NG four times at 10-day intervals (Sharma et al. 1977). Net photosynthetic rate of leaves was reduced 10% by the first spray and 21% by the second; photosynthesis remained steady at the reduced level during the remaining two sprays and final 20 days of the trial. The authors called for additional research to determine the influence on fruit quality and yield (Sharma et al. 1977).

Phytotoxic effects of dicofol products on crop species seem either to be of minor economic consequence or to occur only when the chemical is used several times per year. Multiple applications are now generally avoided because they quickly produce resistant populations of mites. Possible phytotoxic effects of dicofol on natural plant species and plant communities have not been studied.

Invertebrates

Dicofol products kill mites of numerous species that are pests on domestic plants. Other research has shown that nontarget species may also be killed. The LC50 values for six aquatic species are summarized in Table 5. In interpreting results of such tests, note that the shorter the test exposure interval the higher the concentration of toxicant needed to kill 50% of the organisms of a particular species.

In addition to measuring acute toxicities of the estuarine or grass shrimp, *Crangon franciscorum* (Table 5), Sharp et al. (1979) demonstrated that Kelthane-NG had serious effects on these shrimp at dicofol concentrations in water far below LC50 levels measured for this species. These crustaceans experienced 50% mortality after 264 h (11 days) exposure to only 14 parts per billion (ppb) dicofol. Also, rates of feeding, cannibalism, molting, and respiration decreased, generally in a dose–response way, at exposures of 14, 33, and 81 ppb; swimming and escape behavior were abnormal in all shrimp exposed to Kelthane-NG. Khorram and Knight (1977) found that 100 ppb dicofol was sufficient to kill 50% of *C. franciscorum* in 80 h (3.3 days).

The upper chronic limit (lowest concentration of dicofol in water that caused significant mortality, compared with controls in 28 days of exposure) for juvenile amphipods (*Hyalella azteca*) was only 33 ppb (Spehar et al. 1982).

Rawash et al. (1975) determined that waterfleas (*Daphnia magna*), a nontarget species, were killed by lower concentrations of Kelthane-NG than larval mosquitoes (*Culex pipiens*; Table 5); DDT was about 13 times more toxic than Kelthane-NG to *D. magna* (DDT 26-h LC50 = 6.1–6.4 ppb), but Kelthane-NG was twice as toxic as DDT to *C. pipiens* (DDT 24-h LC50 = 230–520 ppb).

Brocksen and Knight (1973), working with *C. franciscorum* and the mysid shrimp, *Neomysis intermedia* (Table 5), considered dicofol "extremely toxic" to these two estuarine species. These authors also reported that San Francisco Bay waters contained as high as 30 ppb dicofol and that concentrations could be higher in the Sacramento–San Joaquin Delta. Thus, concentrations in some natural waters may exceed levels found to have serious toxic effects in the laboratory.

Data from Johnson and Finley (1980) indicated that the stonefly (*Pteronarcys californica*; Table 5) was less sensitive to dicofol product-NG than were young channel catfish (*Ictalurus punctatus*), bluegills (*Lepomis macrochirus*), largemouth bass (*Micropterus salmoides*), cutthroat trout (*Salmo clarki*), or lake trout (*Salvelinus namaycush*; Table 6). The amphipod (*H. azteca*), however, was roughly similar to the fathead minnow in its survival rates when exposed to several concentrations of Kelthane-NG (Spehar et al. 1982), and the striped bass (*Morone saxatilis*) was clearly less sensitive than were the grass or mysid shrimp (Brocksen and Knight 1973; Tables 4 and 5). Tests by the Federal Water Pollution Control Administration (FWPCA 1968) showed that small rainbow trout (*Oncorhynchus mykiss*) were more sensitive to Kelthane-NG than were stoneflies or waterfleas (Tables 4 and 5). Other LC50 tests indicated that DDT was 160 times or 93 times more toxic than Kelthane-NG or dicofol to stoneflies (FWPCA 1968; Johnson and Finley 1980).

Table 5. *Tests of acute toxicity of dicofol products to aquatic invertebrate animals.*

Species	Test system	Chemical[a]	Time of test	LC50[b] in ppb (95% C.I.)	Reference
Crangon franciscorum Grass shrimp	Nonrecirculating, pulse-flow, estuarine	Kelthane	72 h	1,380[c]	Sharp et al. (1979)
Grass shrimp	Nonrecirculating, pulse-flow, estuarine	Kelthane	96 h	508[c]	Sharp et al. (1979)
Grass shrimp	Static, estuarine	Kelthane (80%)	24 h	1,288[c] (777–2,138)	Khorram and Knight (1977)
Grass shrimp	Static, estuarine	Kelthane (80%)	48 h	603[c] (437–832)	Khorram and Knight (1977)
Grass shrimp	Static, estuarine	Kelthane	48 h	350[c]	Brocksen and Knight (1973)
Neomysis intermedia Mysid shrimp	Static, estuarine	Kelthane	48, 96 h	60–65[c]	Brocksen and Knight (1973)
Culex pipiens Mosquito (4th instar larvae)	Static, freshwater	Kelthane	24 h	170–240[d]	Rawash et al. (1975)
Daphnia magna Waterflea	Static, freshwater	Kelthane	26 h	71–90[d]	Rawash et al. (1975)
Waterflea	Static, freshwater	Kelthane	48 h	390[de]	FWPCA (1968)
Pteronarcys california Stonefly	Static, freshwater	Dicofol (100%)	96 h	650[d] (280–1,360)	Johnson and Finley (1980)
Pteronarcys california Stonefly	Static, freshwater	Kelthane	48 h	3,000[d]	FWPCA (1968)
Tubifex tubifex Tubificid worm	Static, freshwater	Kelthane	72 h	500[d]	Naqvi and Ferguson (1968)

[a] Percent of DDTr in original dicofol product not given in any of these studies. Percent of active ingredient is provided in parentheses if given in the publication.
[b] Concentration of active ingredient in water lethal to 50% of the organisms exposed, expressed as parts per billion (ppb).
[c] Based on chemical analyses of water for dicofol.
[d] Based on nominal Kelthane or dicofol concentrations.
[e] Endpoint was immobility, rather than death.

Kelthane-NG was 6 times more toxic than DDT to tubificid worms (*Tubifex tubifex*) in laboratory tests (Naqvi and Ferguson 1968). However, because this population of worms had long been exposed to drainage from cotton agriculture in which dicofol products had not been extensively used (Boyd and Ferguson 1964), they may have been resistant to DDT but not to dicofol products. Under these same agricultural conditions, the mosquitofish had developed resistance to DDT but not to Kelthane-NG (Boyd and Ferguson 1964).

Among terrestrial invertebrates, dicofol toxicity has been tested on three species of bees, including the honey bee. Toxicant solutions applied directly to anesthetized bees produced LD50's (dose, in µg per bee, lethal to 50% of bees exposed) for Kelthane-NG of 78 µg per bee for the alfalfa leafcutting bee (*Megachile rotundata*) and 12 µg per bee for the honey bee (Johansen et al. 1963). Comparable values for DDT were 4.2 and 6.4 µg, thus DDT was more toxic than Kelthane-NG by factors of 19 and 2, respectively. Malathion was considerably more toxic, with LD50 values of 0.23 and 0.46 µg for leafcutting bees and honey bees, respectively.

When alkali bees (*Nomia melanderi*), alfalfa leafcutting bees, and honey bees were caged with alfalfa sprayed 1 h earlier with various chemicals, Kelthane-NG killed 23, 0, and 6% of each species, respectively

(Johansen and Eves 1967). Naled, the most toxic chemical, killed 95–100% of each species. Both Kelthane-NG and naled were applied at 1.1 kg/ha.

When honey bee hives were sprayed directly with pesticides (113 g of 1% solution per hive), Kelthane-NG sprayed on two hives did not kill any bees, whereas carbaryl killed an average of 15% in three hives, and carbofuran, diazinon, and malathion each killed 100% in single hives (Keener and Pless 1974).

Tests of contact and of oral toxicities of 60 pesticides to honey bees resulted in dicofol product-NG LD50 values of >50 and >10 µg per bee, respectively (Stevenson 1978). Comparable values for DDT were 3.9 and 3.7 µg per bee, thus DDT was >13 times and >3 times more toxic than dicofol product. Several other compounds tested <0.1 µg per bee.

Koch et al. (1969) found that Kelthane-NG was stronger than DDT, DDD, DDE, chlordane, or lindane as an inhibitor of $Na^+ - K^+$ATPase and Mg^{++}ATPase enzymes in tissues of the American cockroach (*Periplaneta americana*) and the honey bee.

Moore (1986) cited expert opinion that one application of dicofol product reduced nymphs of predatory thrips about 18%, reduced predatory mites in spring, and had little or no effect on predatory bugs (Hemiptera). The authority, T. Wilson, also believed that both sulfur and propargite, possible alternatives on cotton, would have greater negative effects than dicofol products on populations of predatory insects and predatory mites (Moore 1986).

The EPA concluded that dicofol products are highly toxic to aquatic organisms and impair the reproductive physiology of aquatic invertebrates, but that they are relatively nontoxic to honey bees and alfalfa leafcutting bees (USEPA 1983a).

In summary, the data on toxicity of dicofol products to invertebrates show DDT more toxic (LC50 and dermal LD50 tests) than dicofol product to waterfleas, stoneflies, leafcutting bees, and honey bees; dicofol product was more toxic than DDT only to mosquitoes. Dicofol products were relatively nontoxic to honey bees. In 10 comparisons (LC50 tests or survival rate comparisons) involving six species of aquatic invertebrates and six species of fish, young fish were more sensitive to dicofol products than invertebrates in seven instances, invertebrates were more sensitive in two, and sensitivities were similar in one. Because two species of estuarine invertebrates, a shrimp and an amphipod, experienced significant mortality in laboratory tests at water concentrations of dicofol near or below those reported to exist in San Francisco Bay, effects in the wild are conceivable. The effects of dicofol on most nontarget invertebrates in agroecosystems are poorly known, and its effects on natural invertebrate communities are unstudied.

Vertebrates

Lethal Toxicity

Toxicity tests of dicofol products to vertebrates (Table 6) may be biased by body condition of the test animals. Sensitivity to an organochlorine chemical, as measured by the LC50 (concentration of active ingredient in water that is lethal to 50% of exposed fish, or concentration of active ingredient in a diet that kills 50% of exposed birds) or LD50 (single dose in milligrams of active ingredient per kilogram of bird or mammal that kills 50% of exposed animals), varies with amount of body fat; fat animals of the same species are, on the average, less sensitive than thin ones (Clark 1988).

Trout demonstrated consistently greater sensitivities to dicofol products than other species of fish (Table 6). The comparatively lower toxicity values obtained by McCann (1971a, 1971b) are unexplained but could have resulted if values were based on whole chemical rather than on active ingredients, which were only 35%.

Four species of birds had similar LC50 responses to dicofol products (Table 6). However, three of the species were galliforms, so there was little taxonomic diversity. Note that the toxicity of the dicofol product increased 37% when it was combined with "inert" ingredients in tests of 14-day-old Japanese quail, *Coturnix japonica* (Table 6; Hill and Camardese 1986).

Young laboratory rats (*Rattus norvegicus*) seem more sensitive than older rats, and females may be slightly more sensitive than males; the domestic rabbit (*Oryctolagus cuniculus*) and dog (*Canis familiaris*) seem less sensitive than the laboratory rat (Table 6).

The LD50 for the ring-necked pheasant, *Phasianus colchicus* (Hudson et al. 1984; Table 6), indicates that this bird is far more sensitive to dicofol than are the mammals tested thus far. However, this low LD50 value for the pheasant is questionable because it also indicates that this species is 5 times more sensitive to dicofol than to DDT (DDT LD50 = 1,334 mg/kg; Hudson et al. 1984). Other authors found DDT more toxic than dicofol products to seven of eight species. Johnson and Finley (1980) determined that channel catfish, bluegills, and largemouth bass were 17, 60, and 263 times more sensitive to DDT than to dicofol products (LC50 in water

Table 6. *Tests of acute and subacute toxicity of dicofol products to vertebrate animals.*

Species	Size or age of test animals	Chemical[a]	Time of test	Toxicity (95% C.L.)	Reference
		Fish (LC50[b] as ppb in water[c]			
Cyprinus carpio Common carp	10–12 g	Kelthane	96 h	4,000[d]	Prokopenko et al. (1980)
Pimephales promelas Fathead minnow	31–32 day-old larvae	Kelthane (87.3%)	96 h	510[d] (488–533)	Holcombe et al. (1982)
Gambusia affinis Mosquitofish	Medium-sized fish	Kelthane	24 h	1,900[e]	Boyd and Ferguson (1964)
Lepomis macrochirus Bluegill	1.2 g	Kelthane (35%)	24 h	3,850[e] (3,420–4,330)	McCann (1971a)
Bluegill	1.2 g	Kelthane (35%)	48 h	3,350[e] (2,880–3,890)	McCann (1971a)
Bluegill	1.2 g	Kelthane (35%)	96 h	3,100[e] (2,650–3,630)	McCann (1971a)
Bluegill	—	Dicofol	96 h	510[e]	Johnson and Mayer (1972)
Bluegill	Fingerling	Dicofol (100%)	96 h	520[e] (42–642)	Johnson and Finley (1980)
Micropterus salmoides Largemouth bass	—	Dicofol	96 h	450[e]	Johnson and Mayer (1972)
Largemouth bass	Fingerling	Dicofol (100%)	96 h	395[e]	Johnson and Finley (1980)
Morone saxatilis Striped bass	—	Kelthane	48 h	2,750[d]	Brocksen and Knight (1973)
Ictalurus punctatus Channel catfish	—	Dicofol	96 h	300[e]	Johnson and Mayer (1972)
Channel catfish	Fingerling	Dicofol (100%)	96 h	360[e] (290–447)	Johnson and Finley (1980)
Oncorhynchus mykiss Rainbow trout	2 g	Kelthane (35%)	24 h	1,580[e] (1,230–2,050)	McCann (1971b)
Rainbow trout	2 g	Kelthane (35%)	48 h	1,090[e] (850–1,395)	McCann (1971b)
Rainbow trout	2 g	Kelthane (35%)	96 h	950[e] (743–1,215)	McCann (1971b)
Rainbow trout	—	Dicofol	24 h	110[e]	Johnson and Mayer (1972)
Rainbow trout	50 mm	Kelthane	48 h	100[e]	FWPCA (1968)
Rainbow trout	Juvenile	Kelthane (87.3%)	24 h	335[d] (307–365)	Holcombe et al. (1982)
Rainbow trout	Juvenile	Kelthane (87.3%)	48 h	309[d] (268–357)	Holcombe et al. (1982)
Rainbow trout	Juvenile	Kelthane (87.3%)	72 h	262[d] (232–296)	Holcombe et al. (1982)
Rainbow trout	Juvenile	Kelthane (87.3%)	96 h	210[d] (181–244)	Holcombe et al. (1982)
Salmo clarki Cutthroat trout	Fingerling	Dicofol (74.4%)	96 h	53[e] (41–68)	Johnson and Finley (1980)
Salvelinus namaycush Lake trout	Fingerling	Dicofol (74.4%)	96 h	87[e] (53–142)	Johnson and Finley (1980)

Table 6. *Continued.*

Species	Size or age of test animals	Chemical[a]	Time of test	Toxicity (95% C.I.)	Reference
		Birds (LC50[f] as ppm dry weight in diet)			
Anas platyrhynchos Mallard	10 days old	Dicofol	8 days	1,651[e] (1,356–2,029)	Hill et al. (1975)
Phasianus colchicus Ring-necked pheasant	16 days old	Dicofol	8 days	2,126[e] (1,892–2,387)	Hill et al. (1975)
Colinus virginianus Northern bobwhite	15 days old	Dicofol	8 days	3,010[e] (2,635–3,424)	Hill et al. (1975)
Coturnix japonica Japanese quail	12 days old	Dicofol	8 days	1,418[e] (1,232–1,628)	Hill et al. (1975)
Japanese quail	14 days old	Dicofol (75%)	8 days	1,535[e] (1,201–1,962)	Hill and Camardese (1986)
Japanese quail	14 days old	Kelthane (18.5%)	8 days	1,027[e] (852–1,259)	Hill and Camardese (1986)
		Birds (oral LD50[g] as mg/kg of bird)			
Ring-necked pheasant (males)	4 months old	Dicofol (87.8%)	14 days	265[e] (211–334)	Hudson et al. (1984)
		Mammals (oral LD50[g] as mg/kg of mammal)			
Rattus norvegicus Laboratory rat	—	Kelthane	—	730±90[e,h]	Barker and Maughan (1956)
Laboratory rat (males)	220 g	Kelthane (84.8%)	7 days	1,495[e] (1,039–2,150)	Brown et al. (1969)
Laboratory rat (males)	≥90 days old	Kelthane	12 days	1,100[e] (909–1,331)	Gaines (1960)
Laboratory rat (females)	≥90 days old	Kelthane	18 days	1,000[e] (820–1,220)	Gaines (1960)
Laboratory rat (males)	Young, 140 g	Kelthane (80%)	5 days	809±33[e,h]	Smith et al. (1959)
Laboratory rat (females)	Young, 140 g	Kelthane (80%)	5 days	684±16[e,h]	Smith et al. (1959)
Oryctolagus cuniculus Domestic rabbit (males)	Young, 2 kg	Kelthane (80%)	4 days	1,810±350[e,h]	Smith et al. (1959)
Canis familiaris Domestic dog	—	Kelthane (80%)	—	>4,000[e]	Smith et al. (1959)
		Mammals (dermal LD50[i] as mg/kg of mammal)			
Laboratory rat (males)	≥90 days old	Kelthane	13 days	1,230[e] (1,060–1,427)	Gaines (1960)
Laboratory rat (females)	≥90 days	Kelthane	14 days	1,000[e] (800–1,250)	Gaines (1960)

[a] Percent of DDTr in original dicofol product not given in any of these studies. Percent of active ingredient is provided in parentheses if given in the publication.
[b] Concentration of active ingredient in water lethal to 50% of the animals exposed.
[c] All tests with fish were in static water except those of Holcombe et al. (1982), which used a flow-through system.
[d] Based on chemical analyses of water for dicofol.
[e] Based on nominal dicofol or Kelthane concentrations.
[f] Concentration of active ingredient in diet lethal to 50% of the animals exposed.
[g] Oral dose in milligrams of active ingredient per kilograms of animal lethal to 50% of the animals exposed.
[h] Standard error.
[i] Dermal dose in milligrams of active ingredient per kilograms of animal lethal to 50% of the animals exposed.

tests). Hill et al. (1975) found that northern bobwhite (*Colinus virginianus*), Japanese quail, and ring-necked pheasants were 5, 2, and 7 times more sensitive to DDT than to dicofol products; toxicities to mallard were similar (LC50 dietary tests). Gaines (1960) determined that laboratory rats were 8 times (females) or 10 times (males) more sensitive to DDT than to Kelthane-NG (oral LD50 tests).

Spehar et al. (1982) demonstrated that only 39 ppb dicofol in water can kill 50% of fathead minnow larvae in 7–14 days. Because dicofol has been reported at 30 ppb in San Francisco Bay and may occur at higher concentrations in the Sacramento–San Joaquin Delta (Brocksen and Knight 1973), dicofol could be a threat to fish in some areas.

The threat of lethal oral toxicity of dicofol to birds and mammals is probably small. Stickel and Reichel (1980) fed 100 ppm (dry weight) nominal Kelthane (0.4% DDE) to common grackles (*Quiscalus quiscula*) for a month with no demonstrable effect; there was no mortality or loss of flesh or fat. Domestic dogs consumed diets with as much as 300 ppm (dry weight) nominal Kelthane-NG for a year and were unaffected; rats ate up to nominal 500 ppm (dry weight) for 2 years and also were not affected (Smith et al. 1959). Laboratory mice (*Mus musculus*) received dietary concentrations of 120 to 530 ppm (dry weight) dicofol product-NG for 78 weeks (1.5 years); females showed mild weight loss and males had increased tumors but mortality did not increase (Holder 1986).

Physiological, Morphological, and Behavioral Effects

Small common carp (*Cyprinus carpio*) weighing 10–12 g showed plethora of the spleen, enlargement of the gall bladder, and hemorrhagic exudate in the abdominal cavity after 4 days in water containing 1–20 ppm dicofol (Prokopenko et al. 1980). After 15 days exposure to 0.1 ppm, carp showed increased polymorphonuclear white cells and pathomorphological changes in red blood cells. After 30 days exposure to 0.1 ppm, carp also showed depressed peroxidase activity and increased blood neutrophils (Prokopenko et al. 1980).

Fathead minnows did not school normally after 24 h in water containing 0.22 ppm dicofol; they became lethargic after 78 h (Holcombe et al. 1982). Rainbow trout became lethargic and showed flared operculae, gaping mouths, and rapid gill movements after 48 h in water with 0.238 ppm; after 96 h of exposure, all trout had developed a darker body coloration and half had lost their equilibrium (Holcombe et al. 1982).

The EPA concluded that "dicofol impairs the reproductive physiology of fish" (USEPA 1983a).

Dosages of 300 or 900 ppm (dry weight) nominal Kelthane-NG in the diets of two domestic dogs depressed their adrenal production of 17-hydroxycorticosteroids in response to ACTH (Smith et al. 1959). Diets containing 20, 100, 500, or 1,250 ppm (dry weight) nominal Kelthane-NG, when fed to laboratory rats for 3 months, all caused increases in liver-to-body weight ratios. Liver lesions (focal necroses) occurred mostly at dosage levels of 1,250 and 2,500 ppm (Smith et al. 1959). McKinley and Grice (1960) found significantly heavier livers in female rats fed 100 ppm (dry weight) nominal Kelthane-NG for 28 days.

Alekhina and Kuzminskaya (1982) recorded changes in the lactate dehydrogenase system of the heart and liver when rats were dosed orally with Kelthane-NG. Results of experiments by Kuzminskaya et al. (1983) suggested that dicofol accumulates rapidly in liver, heart, and brain tissues of rats exposed daily by gavage to Kelthane-NG, until enough chemical accumulates (3 to 4 months) to induce microsomal monooxygenases, after which residue levels decline because of the high rate of ongoing induction.

Narloch et al. (1987) determined that both dicofol and Kelthane-NG induced hepatic mixed-function oxygenase enzymes in rats at a potency equal to that of DDT. Intraperitoneal injections of dicofol or Kelthane-NG into rats produced dose-related increases in concentrations of microsomal protein and cytochrome P-450, and in microsomal enzyme activities measured in liver tissue (Narloch et al. 1987).

Weight Effects

Growth (measured as weight increase) of young female laboratory rats (initial average weight, 61 g) fed Kelthane-NG for 3 months at nominal 0, 20, 100, 500, 1,250, or 2,500 ppm (dry weight) was inhibited by dosages ≥100 ppm; in males (initial average weight, 67 g) the inhibiting level was ≥1,250 ppm (Smith et al. 1959). In a 2-year study of rats receiving nominal dietary dosage levels of 0, 20, 100, 250, 500, or 1,000 ppm (dry weight), growth of females was inhibited by Kelthane-NG ≥250 ppm and growth of males by ≥500 ppm (Smith et al. 1959). Brown (1972) fed Kelthane-NG at 63.4 mg/kg of body weight per day to laboratory rats; average weight rose from an initial 308 g to 319 g at 7 days, declined slowly to 308 g at 28 days, and then declined rapidly to 257 g at 40 days when the experiment ended.

Female laboratory mice were fed 120 or 240 ppm (probably dry weight) dicofol product-NG for 78 weeks followed by 15 weeks on clean food (Holder 1986). Mice on the higher dosage showed a mild decrease in weight from week 37 to the end, and mice on the lower dosage showed an even smaller weight loss from week 41 to the end.

In summary, the data on toxicity of dicofol products to vertebrates show trout generally more sensitive (LC50 in water tests) to dicofol products than other species of fish. The single available comparison (oral LD50 tests) between vertebrate classes suggested that ring-necked pheasants were several times more sensitive to dicofol than were laboratory rats. DDT was more toxic than dicofol products to channel catfish, bluegill, largemouth bass (LC50 in water tests), bobwhite, Japanese quail (dietary LC50 tests), and laboratory rats (oral LD50 tests); the two chemicals were similarly toxic to mallard (dietary LC50 tests). Ring-necked pheasants were more sensitive to DDT than to dicofol in LC50 tests but more sensitive to dicofol than to DDT in LD50 tests. Dicofol products caused physiological, morphological, or behavioral effects in carp, fathead minnows, domestic dogs, and laboratory rats, and they inhibited growth or caused weight loss in laboratory rats and mice. Because dicofol on leaves of crop plants was measured at 94 ppm (probably wet weight; equivalent dry weight about 280 ppm) and 400 ppm (probably wet weight; equivalent dry weight about 1,200 ppm), it is conceivable that the increases in liver-to-body weight ratio, growth inhibition, and weight loss seen in laboratory rodents could occur in wild rodents. In laboratory tests, larvae of the fathead minnow died in water concentrations of dicofol similar to those reported in San Francisco Bay. Direct lethal oral poisoning of birds or mammals under natural conditions seems unlikely because ingestion of large amounts (≥100 ppm dietary) of dicofol products for long periods (1 month to 2 years) in the laboratory by several species resulted in no mortalities.

Dicofol Products and Wildlife

The threat of dicofol products to wildlife concerns whether the use of dicofol products liberates enough DDTr into the environment to be significant to wildlife. The first question would be how much DDTr is put into the environment by the use of dicofol products containing DDTr; second, does dicofol break down, or is it metabolized, to DDTr? Third, is dicofol, in and of itself, harmful to wildlife? The last two questions are cogent since manufacturers have reduced DDTr in their products to no more than 0.1%, as required by the EPA. This 0.1% level will add only about 3,000 pounds (1,360 kg)/year of DDTr to the U.S. environment, but the propriety of even this addition, which would be geographically limited, has been questioned (Weisskopf 1986).

Effect of DDTr Applied as Part of Dicofol Products

The most recent nationwide survey of organochlorine residues in European starlings (*Sturnus vulgaris*) revealed that the highest DDTr levels were in California, Texas, Arizona, and New Mexico (Bunck et al. 1987). Because California, Texas, Arizona, Florida, and New York (Rohm and Haas Company 1984; Moore 1986) are the States of greatest Kelthane use, this survey data might suggest that use of dicofol products has been responsible for the high concentrations of DDTr reported in other wildlife (Clark and Krynitsky 1983; Hunt et al. 1986; White and Krynitsky 1986) in the Southwest. This hypothesis is clouded because high DDTr residues were not found in Florida wildlife, because reports of such residues from New York are scarce (see Stone and Okoniewski 1988 for review), and because high DDTr residues were found in wildlife from parts of Texas where dicofol products are not used (Hunt et al. 1986; White and Krynitsky 1986). On the gross geographic scale of statewide areas, high levels of DDTr in the Southwest are most likely the result of heavy DDT usage on cotton before the 1972 ban. A general correspondence with areas of dicofol product use would result because dicofol products are applied to cotton, which continues to be grown in most of the same areas where DDT was used.

So, is there specific evidence that dicofol product use has contributed to the problem of environmental DDTr? First, there are the usage data for dicofol products, which show that large environmental inputs of DDTr have occurred. A 10% level of DDTr coupled with an annual U.S. usage of 3 million pounds (1.4 million kg; active ingredient) of dicofol products indicates that about 300,000 pounds (136,000 kg) of DDTr have been released annually. This is only 2.5% of the DDTr released by the annual use of 12 million pounds (5.5 million kg) of DDT before 1972 (Toner 1984), but dicofol product usage is geographically concentrated. The annual releases of DDTr in California, Florida, Texas, and Arizona have been (presumably during 1985)

100,000 pounds (45,400 kg), 85,700 pounds (38,900 kg), 15,400 pounds (6,900 kg), and 12,600 pounds (5,720 kg), respectively (Moore 1986), and usage is also concentrated within States. For example, DDTr from dicofol products added 30,000 pounds (13,600 kg) of DDTr annually to the environment within the range of the California condor (*Gymnogyps californianus*), an endangered species for which DDE-induced eggshell thinning was a concern when individuals still existed in the wild (USEPA 1984).

Second, an EPA computer study modeled cotton agriculture in Kings County, California (USEPA 1984). This study concluded that by the year 2000 the environmental loading of DDTr from former DDT use plus dicofol product use (assuming 10% DDTr) would total 220,000 pounds (99,800 kg) if dicofol product use was ended in 1984, but would be 450,000 pounds (204,000 kg) if dicofol product use continued—doubling environmental residues in soil, water, and biota (USEPA 1984). The EPA also modeled the Arroyo Colorado River drainage of south Texas because of the heavy use of dicofol products (estimated at 100,000 pounds, or 45,400 kg, yearly) for citrus crops (USEPA 1983a). This model predicted that use of dicofol products containing 10% DDTr, excluding DDTr from all other sources, would result in fish with whole-body concentrations of 9 ppm (wet weight) DDTr—levels well above those needed to cause eggshell thinning when eaten by fish-eating birds (USEPA 1983a).

Third, Hunt et al. (1986) said that lack of declines in DDTr in peregrine falcon eggs in central California between 1969 and 1983 indicated continuing input of DDE; they believed that dicofol products had contributed enough DDTr to prevent this decline.

Finally, EPA, using data from the Rohm and Haas Company, showed that in the Arroyo Colorado drainage, DDTr levels had not declined and that a correlation in fact existed between DDTr levels in fish and dicofol product sales (USEPA 1984). These data may be the best evidence that DDTr from dicofol product use can affect residue levels in wildlife.

Assessments and evaluations have been made that reached conclusions counter to the idea that residues from dicofol product use have contributed significantly to DDTr in the environment. The Rohm and Haas Company (1984) appraised the EPA's Arroyo Colorado model and concluded that it overestimated the potential DDTr from dicofol use by 100 times. Mischke et al. (1985) stated that "dicofol usage in California is not a significant source of DDT residues." However, the Natural Resources Defense Council (Moore 1986) believed that Mischke et al. (1985) had not adequately studied the issue.

There also remains the lack of high DDTr in studies of Florida biota. Pickrell et al. (1984:32) analyzed water, soil, and fish samples from areas of high dicofol product use in Florida and concluded that dicofol product use contributed "some" DDTr to the environment. Tissue samples from only two fish (redear sunfish, *Lepomis microlophus*) from one lake were analyzed and found to contain only trace amounts (<0.1 ppm) of DDE (Pickrell et al. 1984). Toner (1984) pointed out that the same monitoring programs that have revealed high residues in wildlife in the Southwest have few collecting localities in Florida and that the localities they do have are in areas distant from the citrus belt. So, high residues could be present but simply not, as yet, measured.

In summary, there is evidence from several sources (dicofol product use data, computer modeling studies of high-use locations, persistence of DDTr in peregrine falcon eggs in California, and a correlation between DDTr levels and dicofol sales in Texas) that dicofol product use has contributed to environmental levels of DDTr. The evidence from dicofol use data and computer models is based on the presence of DDTr at about 10% of active ingredients at time of manufacture, but it does not assume any breakdown or metabolism of dicofol itself to DDTr. Therefore, a reduction of DDTr in dicofol to no more than 0.1% will almost eliminate this source of DDTr unless a significant amount of dicofol itself is broken down or metabolized to DDTr.

Can Dicofol Break Down, or be Metabolized, to DDTr?

Dicofol can be produced by the breakdown or metabolism of DDT. German cockroaches (*Blattella germanica*), American cockroaches, house flies (*Musca domestica*), and fruit flies (*Drosophila melanogaster*) produce an enzyme that can metabolize DDT to dicofol (Agosin et al. 1961; Menzel et al. 1961). Also, Kiigemagi and Terriere (1972) and Lichtenstein et al. (1971) provided evidence that dicofol can result from the breakdown of DDT in soil. However, the reverse of this reaction wherein dicofol breaks down, or is metabolized, to DDTr, or simply to DDE, is of much greater concern because of the known adverse environmental effects of DDTr.

Several investigators described DDE as an end product without first analyzing the constituents of their original dicofol product. Hughes et al. (1969) found DDE

after in vitro degradation of Kelthane-NG. Brown (1972) found DDE in tissues of laboratory rats fed Kelthane-NG and concluded that DDE was a metabolite of dicofol. Similarly, Lykasova and Rabinovich (1985) concluded that Kelthane-NG had been metabolized to DDTr by domestic chickens (*Gallus domesticus*). Even though McKinley and Grice (1960) analyzed their Kelthane-NG and found DDE before feeding it to laboratory rats, they concluded that DDE might be a metabolite of dicofol. McKinney and Fishbein (1972) described theoretical chemical steps by which dicofol might be converted to DDE in nature.

Many studies have shown that DDTr does not result when dicofol is broken down or metabolized. Archer (1974) irradiated dicofol with ultraviolet light and produced only dicofol and DCBP. Brown et al. (1986) demonstrated that dicofol photodecomposes primarily to DCD and DCBP but not to DDE, and that chloro-DDT is easily dechlorinated to DDE. Kelthane (5.4% DDTr) fed to mallard resulted in metabolically formed DDE, but careful analyses showed that it probably was derived from the chloro-DDT component (Risebrough et al. 1986). Schwarzbach et al. (1988a) fed a purified dicofol product (0.08% chloro-DDT as the only DDTr) to ringed turtle-doves (*Streptopelia risoria*) and found that DDE in the egg yolks increased slightly from 0.013–0.080 ppm to 0.036–0.200 ppm (wet weight). These authors concluded that dicofol was not metabolized to DDE (Schwarzbach et al. 1988a). Brown and Casida (1987) injected dicofol, chloro-DDT, or DCD into laboratory mice and also incubated these compounds with rat-liver microsomes. Chloro-DDT was dechlorinated to DDE by both mice and microsomes. Dicofol was dechlorinated to DCD, and both compounds were metabolized to DCBP and DCBH by both systems.

Perhaps the best available evidence for metabolic production of DDE from dicofol comes from studies in which dicofol products were fed to wild species of birds. G. H. Heinz (U. S. Fish and Wildlife Service, Laurel, Maryland, personal communication; USEPA 1984) fed either dicofol product or DDT to 14-day-old northern bobwhite for 6 weeks. Chemical analysis showed that the highest concentration Kelthane-NG diet, nominally 500 ppm (dry weight), contained 340 ppm dicofol and 3 ppm DDE. Livers of quail fed this diet contained an average 27 ppm (wet weight) DDE (range, 9.9–55 ppm). The highest concentration DDT diet, nominally 100 ppm (dry weight), actually contained 56 ppm DDT, or a total DDTr of 71 ppm. Livers of quail fed this diet averaged 11 ppm (wet weight) DDE (range, 6.6–21 ppm). Seemingly livers of quail receiving the DDT diet should have contained more DDE than those on the dicofol diet. One possible explanation for why they did not is that some of the dicofol was metabolized to DDE. However, the chemical analysis of the dicofol diet did not look for other DDTr compounds, such as chloro-DDT, which probably were present. Thus the data from this study are inconclusive. In another study, Stickel and Reichel (1980) fed a nominal 100 ppm (dry weight) Kelthane-NG diet, which an incomplete analysis showed contained 0.4 ppm DDE, to common grackles for a month and found an average 5.2 ppm (wet weight) DDE in the birds' carcasses. This 13-fold increase in DDE was slightly more than the ninefold increase for the quail (Heinz, personal communication), but Stickel and Reichel (1980) concluded that the dietary 0.4 ppm DDE was enough to account for the DDE found in the grackles.

Two studies in which purified dicofol product was fed to birds also reported DDE in tissues. Wiemeyer et al. (1989) fed Kelthane (no DDTr, 0.1% detection limit) of a new formulation in a meat diet at nominal 10 ppm (wet weight) to captive eastern screech-owls and measured residues in their eggs. The diet contained no measurable DDTr (0.05 ppm detection limit). Nevertheless, DDE was found in some eggs at ND (not detected)–0.22 ppm (wet weight). Wiemeyer et al. (1989) stated that enough DDE or chloro-DDT may have been present in the diet at levels below the detection limit to produce these small amounts in the eggs. Beavers et al. (1989) found as much as 2–3 ppm (wet weight) of DDE in carcasses of mallard after feeding them a diet containing 39 ppm (dry weight) dicofol with no measurable DDE (0.1 ppm detection limit). These authors believed dicofol had been metabolized to DDE (Beavers et al. 1989); however, on the basis of the studies discussed earlier, it seems more likely that these DDE residues resulted from DDE at <0.1 ppm or other unmeasured DDTr in the diet.

In summary, DDE residues found in tissues of northern bobwhite, common grackles, mallard, domestic chickens, and laboratory rats after these animals were fed Kelthane, or purified dicofol products, were attributable either to DDE in the original compound or to metabolism of a DDTr, such as chloro-DDT. Studies with ultraviolet irradiation and photodecomposition revealed no breakdown of dicofol to DDE. Purified dicofol injected into laboratory mice, or incubated with rat-liver microsomes, was reduced to DCBP, DCD, and DCBH, but not to DDE. DDE recovered from eggs of dosed turtle-doves and screech-owls was also accounted for by DDE or chloro-DDT in the diet. At present, there is no compelling evidence that dicofol is broken down or metabolized to DDE.

Reproductive Effects of Dicofol Products

Dicofol products have been shown to affect reproduction negatively in captive fish, birds, and mammals under experimental conditions (Table 7).

Spehar et al. (1982) exposed fathead minnow eggs to dicofol at 0, 8.9, 19, 39, 73, or 125 ppb in water. Hatchability was not affected, but the 125 ppb level delayed hatching 1 day and deformed larvae (Table 7).

Most research into reproductive effects has been carried out with birds (Table 7). Spann (1973) fed mallard diets containing nominal 0, 5, or 10 ppm (dry weight; equivalent wet weights = 0, 1.8, or 3.3 ppm) Kelthane-NG for 5 months and collected eggs during the final 2 months. Upon finding no significant differences in rates of egg production, eggshell cracking, embryonation, embryo viability, hatchability, abnormal or dead hatchlings, and survival at 14 days, he concluded that Kelthane-NG had no effect on this species at these dietary concentrations. Eggshell thickness was not examined.

Bennett et al. (1990) fed dicofol (<0.1% DDTr in original product) to unmated laying mallard hens at 0, 3.4, 9.4, 29, or 97 ppm (dry weight; equivalent wet weights = 0, 1.1, 3.1, 9.7, or 32 ppm) in their diets, or DDE at 0, 3.0, 30, or 110 ppm (dry weight; equivalent wet weights = 0, 1.0, 10, or 36 ppm). Dicofol at 29 ppm (dry weight) significantly reduced both shell thickness (5.1%) and shell weight; at 97 ppm (dry weight) it also significantly reduced shell strength. DDE at both 30 and 107 ppm (dry weight) significantly reduced shell thickness, weight, and strength. The 97 ppm (dry weight) dicofol and 110 ppm (dry weight) DDE diets increased the frequencies of cracked or soft-shelled eggs. Egg production was unaffected by both chemicals. All changes by both chemicals were seen after only 1 or 3 days on diet.

Beavers et al. (1989) fed mallard diets with 0, 0.5, 2.5, 9.8, or 39 ppm (dry weight; equivalent wet weights = 0, 0.17, 0.83, 3.3, or 13 ppm) of dicofol (<0.1% DDTr in original Kelthane), or diets with 36 ppm (dry weight; wet weight equivalent = 12 ppm) of DDE. After an initial 8 weeks on dosage, day length was increased to initiate egg laying. The second and fourth eggs of each clutch were collected for measurements; the other eggs remained with the hen for incubation. Dicofol at 39 ppm (dry weight) significantly reduced shell thickness (13.9%) relative to controls; lesser dietary concentrations produced no significant thinning, but eggshell strength was significantly reduced at 9.8 ppm (dry weight) dicofol. Mallard on 39 ppm (dry weight) dicofol showed a significantly increased frequency of cracked eggs. Egg hatchability and survivability of mallard hatchlings were not significantly affected by dicofol; DDE significantly thinned eggshells, increased cracking, and reduced both egg hatchability and survivability of hatchlings.

Schwarzbach et al. (1988a) fed laying ringed turtledoves three diets, 33 ppm (dry weight; equivalent wet weight = 11 ppm) dicofol (0.08% chloro-DDT as only DDTr in original product), 37 ppm DDE (dry weight; equivalent wet weight = 12 ppm), or control, for about 50 days. Both chemicals significantly reduced shell thickness (dicofol by 9.0%) by similar amounts relative to control birds. Thinning was apparent within the first 5 days for both compounds. Both chemicals reduced clutch size; DDE also increased time between clutches. The concentration of dicofol in the egg yolk was correlated with the percent reduction in shell thickness from pretreatment eggs, but the relation may have been dependent on a single data point for statistical significance. Both DDE and dicofol thinned eggshells, but only dicofol increased the frequency of cracked or broken eggs over controls.

Wiemeyer et al. (1989) fed three diets, 9.9 ppm (wet weight) dicofol (3.4% DDTr in original Kelthane—an older formulation), 9.2 ppm (wet weight) dicofol (no detectable DDTr in original Kelthane—a new formulation), or control, to captive eastern screech-owls. Dosage began 11 January 1985; clutches were laid in mid-March to mid-April. Eggshells were significantly thinner (9.0–13.5%) for owls receiving new Kelthane than for controls. Shell weight and shell thickness index (shell weight in milligrams divided by egg length × width in millimeters) were both lowered significantly by both Kelthanes. Other reproductive parameters were unaffected by Kelthane dosage except that, among younger breeders, the percent of eggs that hatched was lower for owls receiving new Kelthane than for controls. In spite of effects on eggshells, Kelthane had only minimal effects on reproduction of screech-owls.

Fry et al. (1988) gavaged American kestrels daily during courtship and egglaying with Kelthane-NG, *p,p'*-dicofol, or DDE at doses equivalent to 1 or 10 ppm (probably wet weight). At the 10 ppm level, all three chemicals thinned eggshells, increased shell breakage, and reduced both fertility and hatching success. The 10 ppm Kelthane-NG caused testicular feminization of male embryos. Doses of all three chemicals at the 1 ppm level also reduced hatching success.

Clark et al. (1990) fed diets containing 0, 0.92, 2.5, 6.9, or 27 ppm (wet weight) dicofol (no detectable DDTr in original Kelthane) to American kestrels. Dosage

Table 7. *Reproductive effects of dicofol products.*

Species	Exposure[a]	Effects	Reference
Pimephales promelas Fathead minnow (eggs)	125 ppb Dicofol (from Kelthane-NG[b], 87%) in water for 4–5 days	Hatching delayed; larvae deformed	Spehar et al. (1982)
Anas platyrhynchos Mallard	29 ppm(Dry weight) dicofol (<0.1% DDTr) in diet for 1 or 3 days	Eggshell thickness and shell weight reduced	Bennett et al. (1990)
Mallard	97 ppm (Dry weight) dicofol (<0.1% DDTr) in diet for 1 or 3 days	Eggshell thickness, weight, and strength reduced, frequency of cracked or soft eggs increased	Bennett et al. (1990)
Mallard	9.8 ppm (Dry weight) dicofol (from Kelthane, <0.1% DDTr) in diet for about 85 days	Eggshell strength reduced	Beavers et al. (1989)
Mallard	39 ppm (Dry weight) dicofol (from Kelthane, <0.1% DDTr) in diet for about 85 days	Eggshell thickness and strength reduced, frequency of cracked eggs increased	Beavers et al. (1989)
Otus asio Eastern screech-owl	9.2 ppm (Wet weight) dicofol (from Kelthane, no DDTr, 90.5%) in diet for 60–100 days before laying	Eggshell thickness reduced, shell weight and thickness indices reduced, egg hatchability reduced	Wiemeyer et al. (1989)
Eastern screech-owl	9.9 ppm (Wet weight) dicofol (from Kelthane, 3.4% DDTr, 96.7%) in diet for 60–100 days before laying	Eggshell weight and thickness indices reduced	Wiemeyer et al. (1989)
Falco sparverius American kestrel	Nominal 10 ppm (probably wet weight) Kelthane-NG	Eggshell thickness, hatching success, and fertility reduced, frequency of shell breakage increased, testes of male embryos feminized	Fry et al. (1988)
American kestrel	Nominal 10 ppm (probably wet weight) *p,p'*-dicofol	Eggshell thickness, hatching success, and fertility reduced, frequency of shell breakage increased	Fry et al. (1988)
American kestrel	Nominal 1 ppm (probably wet weight) Kelthane-NG or *p,p'*-Dicofol	Hatching success reduced	Fry et al. (1988)
American kestrel	2.5, 6.9, or 27 ppm (Wet weight) dicofol (from Kelthane, no DDTr, 100%) in diet for	Eggshell thickness reduced in dose-related manner	Clark et al. (1990)

Table 7. *Continued.*

Species	Exposure[a]	Effects	Reference
	120–175 days before laying		
American kestrel	6.9 ppm (Wet weight) dicofol (from Kelthane, no DDTr, 100%) in diet for 120–175 days before laying	Egg hatchability reduced	Clark et al. (1990)
Streptopelia risoria Ringed turtle-dove	33 ppm (Dry weight) dicofol (0.08% Cl-DDT, 99.49%) in diet for 50 days inclusive of laying	Eggshell thickness reduced, clutch size reduced, frequency of cracked or broken eggs increased	Schwarzbach et al. (1988a)
Mus musculus House mouse	Nominal 500 ppm (dry weight) Kelthane-NG(84.8%) in diet for five generations	In each generation—weight of young at 21 days reduced, percent mortality at both 5 and 21 days increased, and litter size at both birth and 21 days smaller; for all generations combined—fertility, viability, and lactation indices reduced	Brown (1972)
Rattus norvegicus Laboratory rat	Nominal 225 or 500 ppm (dry weight) Kelthane-NG (84.8%) in diet	No young produced	Brown (1972)
Laboratory rat (females, initial weights 100–120 g)	10 mg/kg per day Kelthane-NG (20%) (=estimated 84 ppm dry weight) in diet for 2–3 months before mating	Insemination rate reduced	Trifonova and Gladenko (1980)
Laboratory rat (females, ≥160 g, nulliparous, inseminated)	10 mg/kg per day Kelthane-NG (20%) (=estimated 110 ppm dry weight) in diet for 10 days after insemination	Pregnancy rate reduced	Trifonova and Gladenko (1980)

[a] Percent of DDTr indicates amount in original dicofol product, not amount in diet. Percent of active ingredient is also provided in parentheses if given in the publication.
[b] Amounts of DDTr not given in publication.

began 21 November 1986; clutches were laid from 23 March to 14 May 1987. Mean eggshell thicknesses at 2.5, 6.9, or 27 ppm were all significantly less (8.0, 12.2, or 14.9%) than the control mean, and egg hatchability was reduced by the 6.9 ppm diet. As in screech-owls, in spite of effects on eggshells, Kelthane had only minimal effects on reproduction.

The shell thinning thus far produced experimentally

with dicofol products (see review, Clark et al. 1990) has been less than the 18% associated with population declines of wild raptors (Lincer 1975).

In a five-generation study of laboratory mice, dietary Kelthane-NG at nominal 500 ppm (dry weight) adversely altered reproduction (Table 7), whereas diets with nominal 7, 25, 100, or 225 ppm (dry weight) did not (Brown 1972). At 21 days of age, litter size of mice on the 500 ppm diet averaged (among the five generations) one or two less than controls. At birth, litter size of mice on the 500 ppm diet averaged (among the five generations) one to four less than control mice. Fertility (pregnancies divided by matings × 100), viability (live young at 5 days divided by total young born minus dead at day 1 × 100), and lactation (weaned young divided by total young born minus dead at day 1 × 100) were reduced among mice on the 500 ppm. No developmental abnormalities were found and fetal resorption rates did not differ among groups. Laboratory rats did not produce young when fed Kelthane-NG at 225 or 500 ppm (dry weight), but reproduction was unaffected on diets of 7, 25, or 100 ppm (dry weight) according to Brown (1972; Table 7).

Trifonova and Gladenko (1980) found laboratory rats reproductively sensitive at dietary concentrations estimated at significantly less than 225 ppm (Table 7). In the first of two experiments the authors fed young (initial weights, 100–120 g) female laboratory rats diets with 0, 5, or 10 mg Kelthane-NG/kg of rat per day for 2 months. Then the rats were placed with males three times weekly for a month or until naturally inseminated. Female rats were killed 20 days after insemination. Only 1 of 15 (7%) control rats was not inseminated, whereas 5 of 18 (28%) females that received 5 mg/kg per day and 8 of 19 (42%) females that received 10 mg/kg per day diet were not inseminated. The ratio for the 10 mg/kg per day females was significantly different from controls ($\chi^2 = 5.41$, $P = 0.020$). The proportion of inseminated rats not pregnant was 2 of 14 (14%) controls, 3 of 13 (23%) females fed 5 mg/kg per day, and 4 of 11 (35%) females fed 10 mg/kg per day Kelthane-NG. None of the ratios, separately or combined, was significantly different from controls. The average number of fetuses per pregnant female in the control (8.6) was not significantly different from the average (8.9) in the dosed samples. In the other experiment, inseminated, nulliparous females, weighing at least 160 g were fed 10 mg/kg per day Kelthane-NG for 10 days after insemination and killed 20 days after insemination. Pregnancy was affected; all 13 controls were pregnant compared with only 6 of 12 (50%) dosed rats ($\chi^2 = 8.55$, $P = 0.004$). The numbers of fetuses per pregnant female were similar, 9.1 for controls and 11.5 for dosed rats.

Assuming an average weight of 150 g and an average food intake of 18 g/day for rats in the first experiment of Trifonova and Gladenko (1980), and an average weight of 220 g and an average food intake of 20 g/day for rats in their second experiment, I estimated dietary concentrations of about 42 and 84 ppm (dry weight) in the first experiment and 110 ppm in the second. Therefore, the results of Trifonova and Gladenko (1980) suggest that Kelthane-NG affected estrus and the establishment or maintenance of pregnancy at dietary concentrations that are sometimes available on agricultural crop plants.

Research results allow comparison of effects on bird reproduction from dicofol products with effects from DDE, as well as comparisons among several bird species of reproductive effects from dicofol products. DDE fed to mallard hens at nominal 10 ppm (dry weight) increased eggshell cracking and reduced production of hatchlings (Heath et al. 1969), but Kelthane-NG at this same dietary concentration had no reproductive effects (Spann 1973). DDE at 36 ppm (dry weight) lowered hatching success and survival of mallard hatchlings, but dicofol (<0.1% DDTr in original Kelthane) at 39 ppm (dry weight) did not (Beavers et al. 1989). Schwarzbach et al. (1988a) found that when similar levels of dicofol (33 ppm dry weight; 0.08% chloro-DDT as only DDTr in original dicofol product) or DDE (37 ppm dry weight) were fed to ringed turtle-doves, only DDE increased time between clutches; however, only dicofol increased the incidence of cracked or broken eggs.

In kestrels, DDE at nominal 10 ppm (2.8 ppm equivalent wet weight; Wiemeyer and Porter 1970) and dicofol (no detectable DDTr in original Kelthane) at 2.5 ppm (wet weight; Clark et al. 1990) thinned eggshells significantly. Fry et al. (1988) found that Kelthane-NG, *p,p'*-dicofol, or DDE at nominal 1 ppm (probably wet weight) each reduced hatching success of kestrels; however, Clark et al. (1990) found reduced hatchability of kestrels fed 6.9 ppm but not 0.92 or 2.5 ppm (wet weight). At comparable dietary concentrations, DDE seems to be a stronger shell-thinner than are dicofol products (Clark et al. 1990). All together, dicofol products probably have less severe reproductive effects on birds than does DDE (Clark et al. 1990).

Mallard exhibited an increased frequency of cracked eggshells at 39 ppm (dry weight) dicofol (<0.1% DDTr in original Kelthane) according to Beavers et al. (1989). Similarly, turtle-doves receiving 33 ppm (dry weight) dicofol (0.08% chloro-DDT as only DDTr in original dicofol product) produced more cracked or broken eggs,

but they also showed reduced clutch size (Schwarzbach et al. 1988a). However, screech-owls receiving either 9.2 ppm (wet weight; 28 ppm equivalent dry weight) dicofol (no detectable DDTr in original Kelthane) or 9.9 ppm (wet weight; 30 ppm dry weight equivalent) dicofol (3.4% DDTr in original Kelthane) showed no such effects (Wiemeyer et al. 1989). In kestrels, Fry et al. (1988) found increased shell breakage, reduced fertility, and reduced hatching success at nominal 10 ppm (probably wet weight; 30 ppm equivalent dry weight) of *p,p'*-dicofol or Kelthane-NG. Only reduced hatchability was seen when 6.9 ppm (wet weight; 21 ppm dry weight equivalent) dicofol (no detectable DDTr in original Kelthane) was fed to other kestrels (Clark et al. 1990). Kestrels exhibited more dicofol-induced eggshell thinning than turtle-doves because doves metabolize dicofol more readily (Schwarzbach et al. 1988b). Thus, there seem to be species differences in reproductive sensitivity to dicofol products.

Species differences may also occur in mammals. Kelthane-NG at nominal 500 ppm (dry weight) in the diet of laboratory mice had negative reproductive effects (Brown 1972). However, in laboratory rats a diet with nominal 225 ppm (dry weight) Kelthane-NG prevented reproduction altogether (Brown 1972).

The relevance to fish and wildlife populations of the experimental results summarized here depends on whether sufficient dosages ever occur in nature. Levels in water might reach 125 ppb at sites such as that near Buckeye, Arizona, where sediment contained 199 ppm (wet or dry weight not indicated) of dicofol (Kepner 1986), and so cause reproductive problems such as those seen in fathead minnows. My summary of data on dicofol residues in and on plant materials shows that wild rodents could be exposed to levels in contaminated diets similar to those causing reproductive problems in laboratory rats. However, dicofol residues that were measured in whole, free-living fish, birds, or bird eggs, and so are at positions in food chains where raptorial and fish-eating birds might eat them, have probably been too low (<5 ppm wet weight) to cause reproductive problems (Table 8).

Dicofol Residues in Wildlife

Residue Burdens

Dicofol residues have been found in free-living fish, and in wild birds or their eggs (Table 8). Bird eggs analyzed by S. S. Hurt (Rohm and Haas Company, Spring House, Pennsylvania, personal communication; Table 8) came from localities where dicofol products are heavily used; most contained dicofol residues, thereby confirming that residues readily persist in wildlife. However, White and Krynitsky (1986) measured organochlorine residues in western kingbirds (*Tyrannus verticalis*), house sparrows (*Passer domesticus*), Brazilian free-tailed bats (*Tadarida brasiliensis*), and whiptail lizards (*Cnemidophorus* spp.) in the Rio Grande and Pecos River drainages of New Mexico and Texas. They looked carefully for dicofol and DCBP without finding them, even though DDE in carcasses of these four species reached 47, 35, 46, and 100 ppm, respectively (wet weight). This indicates that dicofol residues in wildlife may be limited primarily to high-use areas.

DDE was generally more abundant than dicofol residues in the same samples. DDE in carp and channel catfish from the Gila River in Arizona measured 0.04–23 ppm and 0.65–6.3 ppm (wet weight; Kepner 1986) exceeding levels of dicofol residues (Table 8) by orders of magnitude. In California, DDE in channel catfish from the Sutter Bypass and from the San Joaquin, Stanislaus, and Tuolumne rivers and in carp from the New River measured 0.41, 2.3, 1.1, 0.64, and 0.90 ppm (wet weight), respectively (Watkins et al. 1985), exceeding dicofol residues (Table 8) by 2 to 6 times.

DDE in red-winged blackbirds from the Gila River also was measured at levels (0.86–41 ppm wet weight; Kepner 1986) several times greater than dicofol residues (Table 8). DDE in eggs of northern mockingbirds (*Mimus polyglottos*) from Florida, screech-owls from Texas, common barn-owls (*Tyto alba*) from Texas, and kestrels from California averaged 0.33, 11, 5.9, and 7.7 ppm (wet weight; Hurt, personal communication), exceeding dicofol residues (Table 8) by 5 to 64 times. However, DDE levels in eggs of northern cardinals, *Cardinalis cardinalis*, (0.20 ppm) and screech-owls from Florida (0.48 ppm; Hurt, personal communication) were similar to those of dicofol residues (Table 8).

The highest dicofol residues found in wildlife thus far were in screech-owl eggs from central Florida (Table 8). Of 15 eggs, 3 contained dicofol residues above 1 ppm. These amounts were 2.2, 2.3, and 4.6 ppm (wet weight), with dicofol itself at 0.82, 0.91, and 1.8 ppm (wet weight). Whether these residues are harmful to reproduction in wild screech-owls is not known. The data of Schwarzbach et al. (1988a) suggest some slight thinning in turtle-dove eggshells with as little as 2 to 5 ppm (wet weight) dicofol (no metabolites) in the egg yolk, but Wiemeyer et al. (1989) showed that captive screech-owls can be heavily dosed with dicofol (9.2 or 9.9 ppm

Table 8. *Residues of dicofol and metabolites in free-living animals.*

Species	Tissue	Residue (ppm wet weight) Dicofol[a]	DCBP	DCD	Locality	Reference
Mugil spp. Mullet	Muscle, viscera[c]	—[b]	—[b]	—[b]	Mediterranean Sea, south Spain	Barbera et al. (1986)
Cyprinus carpio Common carp	Whole body[c]	ND[d]–0.25[e]		—	Lower Gila River, Arizona	Kepner (1986)
Common carp	Muscle[c]	0.19	<0.03	—	New River, California	Watkins et al. (1985)
Ictalurus punctatus Channel catfish	Whole body[c]	ND–0.56[e]		—	Lower Gila River, Arizona	Kepner (1986)
Channel catfish	Muscle[c]	0.19	0.04	—	Sutter Bypass, California	Watkins et al. (1985)
Channel catfish	Muscle[c]	0.48	0.08	—	San Joaquin River, California	Watkins et al. (1985)
Channel catfish	Muscle[c]	0.16	<0.03	—	Stanislaus River, California	Watkins et al. (1985)
Channel catfish	Muscle[c]	0.20	0.04	—	Tuolumne River, California	Watkins et al. (1985)
Otus asio Eastern screech-owl	Egg	0.20[f] (0.04–1.8)	0.09[f] (<0.03–1.2)	0.20[f] (0.04–1.5)	Central Florida	S. S. Hurt (pers. comm.)
Eastern screech-owl	Egg	0.14[f] (0.07–0.26)	0.08[f] (0.04–0.10)	0.17[f] (0.12–0.20)	South Texas	S. S. Hurt (pers. comm.)
Tyto alba Common barn-owl	Egg	—[g] (ND–ND)	—[g] (ND–ND)	—[g] (ND–ND)	San Joaquin Valley, California	S. S. Hurt (pers. comm.)
Common barn-owl	Egg	—[g] (<0.03–<0.03)	—[g] (<0.03–0.04)	0.05[f] (<0.03–0.06)	South Texas	S. S. Hurt (pers. comm.)
Falco sparverius American kestrel	Egg	—[g] (ND–0.04)	—[g] (ND–ND)	—[g] (ND–0.06)	San Joaquin Valley, California	S. S. Hurt (pers. comm.)
Zenaida macroura Mourning dove	Egg	—[g] (ND–0.04)	—[g] (ND–<0.03)	—[g] (ND–0.04)	Central Florida	S. S. Hurt (pers. comm.
Mourning dove	Egg	—[g] (ND–ND)	—[g] (ND–ND)	—[g] (ND–0.03)	San Joaquin Valley, California	S. S. Hurt (pers. comm.)
Mourning dove	Egg	—[g] (ND–0.16)	—[g] (ND–0.07)	—[g] (ND–0.07)	South Texas	S. S. Hurt (pers. comm.)
Cardinalis cardinalis Northern cardinal	Egg	—[g] (<0.03–0.64)	0.05[f] (ND–0.19)	—[g] (ND–0.14)	Central Florida	S. S. Hurt (pers. comm.)
Mimus polyglottos Northern mockingbird	Egg	0.04[f] (ND–0.18)	—[g] (ND–0.05)	—[g] (ND–<0.03)	Central Florida	S. S. Hurt (pers. comm.)
Agelaius phoeniceus Red-winged blackbird	Whole body[c]	ND–0.50[e]		—	Lower Gila River, Arizona	Kepner (1986)

[a] May include DCBP and DCD if separate values not given for these metabolites.
[b] Quantification did not separate dicofol from DDTr.
[c] Composite sample(s).
[d] Not detected in chemical analysis.
[e] Totals for dicofol and DCBP.
[f] Geometric mean and range; values estimated as ND or < detection limit entered mean calculations as one-half the detection limit.
[g] Residues too low to quantify in > half of samples.

wet weight) without harm to reproduction.

Dicofol residues produced under laboratory or pen conditions are much higher than those found in the field (Tables 7 and 8). However, high concentrations such as those accumulated by amphipods and fathead minnows (Table 9) may actually occur in the environment because dicofol has been reported at 30 ppb in San Francisco Bay (Brocksen and Knight 1973), a level similar to the maximum used to produce these residue loads (Table 9). Further up in food chains, raptorial birds have exhibited eggshell thinning when fed dicofol (no detectable DDTr in original Kelthane) at only 2.5 or 9.2 ppm (wet weight; Clark et al. 1990; Wiemeyer et al. 1989)— concentrations well below those produced experimentally in fathead minnows (Table 9).

Species differences may occur in dicofol burdens. Even though the dosage to bobwhite was more than 3 times that to grackles, and feeding intervals were similar, carcasses of grackles contained nearly 4 times more dicofol than those of quail (Table 9). Schwarzbach et al. (1988b) found greater liver accumulation of dicofol in dosed American kestrels than in similarly dosed ringed turtle-doves. Livers of bobwhites contained only DCBP and not dicofol (Table 9). However, this absence of dicofol in the liver may reflect a degradation during the chemical analysis (as occurred with egg yolk; Krynitsky et al. 1988), because dicofol was found in livers of mallard (Table 9) and kestrels (Schwarzbach et al. 1988b).

Birds seem to concentrate DDE in the egg more readily than dicofol. Egg yolks of ringed turtle-doves fed 33 ppm (dry weight) dicofol (0.08% Cl-DDT as only DDTr in original dicofol product) contained their greatest average dicofol residue (14 ppm wet weight) after 42–48 days of dosage (Schwarzbach et al. 1988a; Table 9). By contrast, yolks of two eggs from doves fed 37 ppm DDE (dry weight) for only 13 and 21 days contained 31 and 57 ppm (wet weight) DDE, respectively (Schwarzbach et al. 1988a). Similarly, when Bennett et al. (1990) fed 29 ppm (dry weight) dicofol (<0.1% DDTr in original dicofol product) to mallard, their egg yolks averaged 37 ppm (wet weight) dicofol (Table 9), whereas 30 ppm (dry weight) DDE in the diet produced an average 92 ppm (wet weight) DDE in the same interval.

Studies of dicofol in laboratory rats (Table 9) show a tendency for females to accumulate more residues in their fat than males do. Carcasses of female screech-owls contained more dicofol than did those of males (Table 9), but Wiemeyer et al. (1989) believed the difference could not be attributed to sex because of other complicating factors. The data of Beavers et al. (1989) showed no obvious sex difference in dicofol residues.

Uptake and Loss

Eaton et al. (1983) estimated uptake rates of dicofol (from Kelthane, 0.35% DDE) for fathead minnows in 28-day tests. Testing was done at a "high" concentration of dicofol in water (12–15 ppb) and at a "low" concentration (1.2–1.3 ppb). Experimental addition of suspended clay to the water reduced both rate of dicofol uptake and total amount taken up by the fish. Ninety-five percent of steady-state concentrations were estimated to be reached in 39–47 days at the high dicofol concentration and in 60 days at the low concentration (Eaton et al. 1983).

Dicofol residues in carcasses of mallard that were dosed (<0.1% DDTr in original Kelthane) for 18 weeks peaked after 10 to 15 weeks; residues declined rapidly after dosage with an estimated half-life of 17 to 20 days (Beavers et al. 1989).

Ringed turtle-doves were fed dicofol (0.08% Cl-DDT as only DDTr in original dicofol product) and their egg yolks were analyzed in samples representing 13–16, 21–28, 29–39, and 42–48 days on dosage. Dicofol increased in each successive sample; DCBP seemed to plateau in the final two periods (Schwarzbach et al. 1988a).

Lykasova and Rabinovich (1985) gave chickens a single dose of 800 mg Kelthane-NG/kg by stomach tube and took blood samples at 1, 3, 5, 7, and 12 h and at 1, 2, 3, and 14 days. Dicofol peaked at 12 h in plasma, but by 36 h it had declined about 80% and remained at this low level throughout the experiment. Dicofol in feces increased more slowly, peaking at 48–72 h, then declining to zero at 14 days.

Smith et al. (1959) fed laboratory rats Kelthane-NG at nominal 32 ppm (dry weight) for 12 weeks, followed by clean food for 14 weeks. Dicofol concentrations were measured at 8, 10, 12, 14, 16, 18, 20, and 26 weeks. Dicofol in fat of females rose throughout dosing and peaked at 12 weeks. Dicofol in males peaked at 8 weeks but remained nearly as high at 10 and 12 weeks. At 14 weeks, amounts dropped 42% in females and 74% in males. By 26 weeks, amounts in females had declined 92% from their peak; amounts in males had reached zero.

Brown et al. (1969) gave rats single intraperitoneal injections of Kelthane-NG at 230 mg/kg and took tissue samples at 2, 4, 8, 16, 32, 40, 48, and 96 h. Concentrations of both dicofol and DCBP peaked at 40 h in blood, brain, kidney, liver, muscle, and lung. By 96 h, declines in dicofol in these tissues were 60, 81, 63, 50, 37, and 70%, respectively; declines in DCBP were

64, 75, 62, 48, 36, and 69%. Concentrations of dicofol and DCBP in heart and testes peaked at 32 h, with declines by 96 h of 69 and 25% in dicofol and 67 and 27% in DCBP. Residues in fat and feces continued to rise throughout sampling. Residues in urine were elevated from 4 h onward, with a slight peak at 32 h. Brown et al. (1969) gavaged laboratory rats daily for 40 days with 75 mg/kg Kelthane-NG and sampled at 15, 29 or 30, and 40 days. In the liver, concentrations of both dicofol and DCBP were highest at 15 days followed by identical 87% declines by 40 days. Concentrations in blood were highest at 29 days but with only minimal fluctuations among samples. Concentrations of dicofol and DCBP in both urine and feces were highest in the final sampling interval of 36–40 days.

Kuzminskaya et al. (1983) gave laboratory rats daily gavage dosages of 14 mg/kg Kelthane-NG for 9 months and sampled at 1, 3, 6, and 9 months. Concentrations of dicofol in both heart and brain peaked at 3 months and declined an identical 71% by 9 months. Concentrations in the liver peaked at 6 months and declined 59% by 9 months.

Dicofol in butterfat from three cows that were fed nominal 2.0 ppm (dry weight) Kelthane-NG for 76 days dropped to zero after 25 days on uncontaminated feed (Zweig et al. 1963). These cows were then fed 1.0 ppm Kelthane-NG for 42 days. Concentrations in butterfat plateaued in about 9 days at both dosages.

In summary, dicofol residues have been found in free-living fish, and in wild birds or their eggs, but concentrations were low (<5 ppm wet weight). DDE residues in the same samples were usually much higher. Given that fathead minnows accumulated 81 ppm (wet weight) dicofol from water containing 19 ppb, and that 30 ppb dicofol was reported in water from San Francisco Bay, and that raptorial birds have shown eggshell thinning when fed 2.5 and 9.2 ppm (wet weight) dicofol, a risk to fish-eating birds at some locations is conceivable. The low residue concentrations of dicofol found thus far in wildlife in relation to the high residues accumulated in laboratory and pen studies suggest that amounts added to the environment have been relatively small, or that dicofol is readily broken down in natural food chains, or that appropriate samples have yet to be analyzed. Residue data from birds indicate that DDE is accumulated more readily than dicofol and that some species may accumulate dicofol more readily than others. Uptake and loss rates for dicofol probably vary with species, method and amount of dosage, and tissue examined.

Conclusions and Recommendations

The legal sale of dicofol products containing more than 0.1% DDTr ceased 1 January 1989, and the legal use of such products ended 31 March 1989 (Moore 1986). New formulations of Kelthane analyzed at Patuxent Wildlife Research Center in 1986–87 contained no detectable DDTr (Clark et al. 1990; Wiemeyer et al. 1989). Unfortunately, the effect of DDTr added to the U.S. environment by dicofol products from 1957 through 1988 will probably never be accurately evaluated, but this addition should now be ended and concern has dissipated.

Continued concern for wildlife over the addition of environmental DDTr could be based on breakdown or metabolism of dicofol to DDTr. However, several careful studies (Archer 1974; Brown et al. 1986; Risebrough et al. 1986; Brown and Casida 1987; Schwarzbach et al.

Table 9. *Residues of dicofol and metabolites in captive, dosed animals.*

Species	Tissue	Residue (ppm wet weight)			Exposure[b]	Reference
		Dicofol[a]	DCBP	DCD		
Hyalella azteca Amphipod (juveniles)	Whole animal	33[c], 100[c], or 230[c]	—	—	5.3, 9.4, or 19 ppb Dicofol (from Kelthane-NG[d], 87%) in water for about 30 days	Spehar et al. (1982)
Pimephales promelas Fathead minnow (larvae)	Whole animal	28[c], 81[c], or 230[c]	—	—	8.9, 19, or 39 ppb Dicofol (from Kelthane-NG, 87%)	Spehar et al. (1982)

Table 9. *Continued.*

Species	Tissue	Residue (ppm wet weight) Dicofol[a]	DCBP	DCD	Exposure[b]	Reference
					in water for about 30 days	
Anas platyrhynchos Mallard	Carcass[c]	0.30[f], 2.5[f], or 39[f]	<0.1[f], <0.1[f], or 0.31[f]	<0.1[f], <0.1[f], or 0.50[f]	0.51, 2.5, or 39 ppm (Dry weight) dicofol (from Kelthane, <0.1% DDTr) in diet for 126 days	Beavers et al. (1989)
Mallard	Liver	<0.1[f], 0.31[f], or 2.5[f]	<0.1[f], <0.1[f], or <0.1[f]	<0.1[f], 0.32[f], or 4.6[f]	0.51, 2.5, or 39 ppm (Dry weight) dicofol (from Kelthane, <0.1% DDTr) in diet for 126 days	Beavers et al. (1989)
Mallard	Whole egg contents	0.36[f], 1.6[f], or 28[f]	ND[g], <0.1[f], or 0.44[f]	ND, <0.1[f], or 0.48[f]	0.51, 2.5, or 39 ppm (Dry weight) dicofol (from Kelthane, <0.1% DDTr) in diet for about 85 days	Beavers et al. (1989)
Mallard	Hatchling, 14 days old	0.13[f], 0.68[f], or 11[f]	ND, <0.1[f], or 0.55[f]	<0.1[f], 0.11[f], or 2.2[f]	0.51, 2.5, or 39 ppm (Dry weight) dicofol (from Kelthane, <0.1% DDTr) in diet for 14 days	Beavers et al. (1989)
Mallard (females)	Fat	17±5[h,i], 50±5[h,i], 140[i], or 450[i]	—	—	3.4, 9.4, 29, or 97 ppm (Dry weight) dicofol (<0.1% DDTr) in diet for 42 days	Bennett et al. (1990)
Mallard (females)	Liver	1.6±0.8[h,i], 5.6±1.7[h,i], 9.5[i], or 14[i]	—	—	3.4, 9.4, 29, or 97 ppm (Dry weight) dicofol (<0.1% DDTr) in diet for 42 days	Bennett et al. (1990)
Mallard	Egg yolk	5.4±0.3[h,i], 18±1[h,i], 37±2[h,i], 150±10[h,i]	—	—	3.4, 9.4, 29, or 97 ppm (Dry weight) dicofol (<0.1% DDTr) in diet for 37–42 days	Bennett et al. (1990)
Otus asio Eastern screech-owl (males)	Carcass	3.4[j] (2.2–4.6)	0.92[j] (0.42–1.2)	2.8[j] (1.7–3.4)	9.2 or 9.9 ppm (Wet weight) dicofol (from	Wiemeyer et al. (1989)

Table 9. *Continued.*

Species	Tissue	Residue (ppm wet weight) Dicofol[a]	DCBP	DCD	Exposure[b]	Reference
					Kelthane, 90.5 or 96.7%, no DDTr or 3.4% DDTr) in diet for 104 days	
Eastern screech-owl (females)	Carcass	6.2[j] (4.5–7.8)	1.5[j] (1.1–2.2)	4.4[j] (3.4–6.6)	9.2 or 9.9 ppm (Wet weight) dicofol (from Kelthane, 90.5 or 96.7%, no DDTr or 3.4% DDTr) in diet for 104 days	Wiemeyer et al. (1989)
Eastern screech-owl (females)	Liver	0.33[j] (ND–1.6)	ND	0.64[j] (ND–1.8)	9.2 or 9.9 ppm (Wet weight) dicofol (from Kelthane, 90.5 or 96.7%, no DDTr or 3.4% DDTr) in diet for 104 days	Wiemeyer et al. (1989)
Eastern screech-owl	Entire egg contents	3.2	1.2	2.2	9.2 ppm (Wet weight) dicofol (from Kelthane, 90.5%, no DDTr) in diet for 104 days	Krynitsky et al. (1988)
Gallus domesticus Domestic chicken (hens)	Fat, white meat, or dark meat	28[c], 2.1[c], or 4.2[c]	—	—	Capsule dose equal to nominal 10–15 ppm (dry weight) Kelthane-NG (85%) in diet for 5 days	McCaskey et al. (1968)
Domestic chicken	Egg yolk	14[k]	—	—	Capsule dose equal to nominal 10–15 ppm (dry weight) Kelthane-NG (85%) in diet for 5 days	McCaskey et al. (1968)
Domestic chicken (6 months old)	Blood plasma	1.3[cl]	—	—	Single gavage dose 800 mg/kg Kelthane-NG (20%)	Lykasova and Rabinovich (1985)
Domestic chicken (6 months old)	Fat, muscle, liver, and kidney	22[c,m], 0.4[c,m], 3.0[c,m], and 5.6[c,m]	—	—	Single gavage dose 800 mg/kg Kelthane-NG (20%)	Lykasova and Rabinovich (1985)

Table 9. *Continued.*

Species	Tissue	Residue (ppm wet weight) Dicofol[a]	DCBP	DCD	Exposure[b]	Reference
Colinus virginianus Northern bobwhite (14 days old)	Carcass	22[c] (17–28)	ND	—	341 ppm (Dry weight) dicofol (from Kelthane-NG) in diet for 42 days	Heinz (personal communication); USEPA (1984)
Northern bobwhite (14 days old)	Liver	ND	0.04[c], 0.18[c], 0.10[c], 3.9[c], or 3.5[c]	—	12, 35, 93, 207, or 341 ppm (Dry weight) dicofol (from Kelthane-NG) in diet for 42 days	Heinz (personal communication); USEPA (1984)
Streptopelia risoria Ringed turtle-dove	Egg yolk	14[c,k]	9.9[c,k]	—	33 ppm (Dry weight) dicofol (0.08% Cl-DDT) in diet for about 50 days	Schwarzbach et al. (1988a)
Quiscalus quiscula Common grackle (males)	Carcass	83[c] (59–109)	—	—	Nominal 100 ppm (dry weight) Kelthane (0.4% DDE) in diet for 31 days	Stickel and Reichel (1980)
Rattus norvegicus Laboratory rat (males)	Fat	26[c,n]	—	—	Nominal 32 ppm (dry weight) Kelthane-NG (80%) in diet for 12 weeks	Smith et al. (1959)
Laboratory rat (females)	Fat	74[c,o]	—	—	Nominal 32 ppm (dry weight) Kelthane-NG (80%) in diet for 12 weeks	Smith et al. (1959)
Laboratory rat (males)	Fat	0.69[c], 2.7[c], or 14[c]	—	—	Nominal 1, 3.2, or 10 ppm (dry weight) Kelthane-NG (80%) in diet for 10 weeks	Smith et al. (1959)
Laboratory rat (females)	Fat	2.2[c], 7.2[c], or 31[c]	—	—	Nominal 1, 3.2, or 10 ppm (dry weight) Kelthane-NG (80%) in diet for 10 weeks	Smith et al. (1959)
Laboratory rat (males)	Fat	36[c], 44[c], 140[c], 280[c], or 540[c]	—	—	Nominal 20, 100, 250, 500, or 1,000 ppm (dry weight) Kelthane-NG	Smith et al. (1959)

Table 9. *Continued.*

Species	Tissue	Residue (ppm wet weight) Dicofol[a]	DCBP	DCD	Exposure[b]	Reference
					(80%) in diet for 2 years	
Laboratory rat (females)	Fat	70[c], 120[c], 410[c], 870[c], or 1,500	—	—	Nominal 20, 100, 250, 500, or 1,000 ppm (dry weight) Kelthane-NG (80%) in diet for 2 years	Smith et al. (1959)
Laboratory rat (males)	Blood	2.6[c,p]	0.7[c,p]	—	Daily gavage dose of 75 mg/kg Kelthane-NG (84.8%)for 40 days	Brown et al. (1969)
Laboratory rat (males)	Liver	66[c,q]	13[c,q]	—	Daily gavage dose of 75 mg/kg Kelthane-NG (84.8%) for 40 days	Brown et al. (1969)
Laboratory rat (males)	Fat, muscle, brain, or kidney	69[c,r], 17[c,r], 8.1[c,r], or 9.1[c,r]	21[c,r], 3.1[c,r], 1.4[c,r], or 1.4[c,r]	—	Daily gavage dose of 75 mg/kg Kelthane-NG (84.8%) for 40 days	Brown et al. (1969)
Laboratory rat	Liver, brain, or heart	4.7[s], 4.4[t], 9.6[t]	—	—	Daily gavage dose of 14 mg/kg Kelthane-NG for 9 months	Kuzminskaya et al. (1983)
Bos taurus Domestic cow	Butterfat or omental fat[u]	0.34[c], or 1.1 and 2.7	—	—	Nominal 2.0 ppm (dry weight) Kelthane-NG in diet for 76 days	Zweig et al. (1963)

[a] May include DCBP and DCD if separate values not given for these metabolites.
[b] Percent of active ingredient is provided in parentheses if given in the publication.
[c] Mean and (range), if range given.
[d] Amounts of DDTr not given in publication.
[e] Whole animal minus skin, feet, beak, brain, and gastrointestinal tract.
[f] Peak values after various dosage intervals.
[g] Not detected in chemical analysis.
[h] Mean and standard error.
[i] Values are p,p′-dicofol only.
[j] Geometric mean and range.
[k] Lower values were obtained for shorter exposure periods.
[l] Peak value 12 h after dosage.
[m] Values are 24 h after dosage.
[n] Peak value after 8 weeks on dosage.
[o] Peak value after 12 weeks on dosage.
[p] Peak reached on day 29 of dosage.
[q] Peak reached on day 15 of dosage.
[r] Concentrations on day 40. Authors also present data for liver, heart, testes, and lung on day 40.
[s] Peak concentration reached after 6 months of dosage.
[t] Peak concentrations reached after 3 months of dosage.
[u] Samples collected 28 days after 2.0 ppm dosage ended.

1988a; Wiemeyer et al. 1989) produced no evidence that this transformation occurs, and most remaining concern is focused on the toxic effects of dicofol itself on wildlife, and on the behavior and fate of dicofol in ecosystems.

Present information on residue loadings, acute toxicity, and reproductive effects in birds maintains that dicofol is, at most, a minor environmental concern. Amounts of dicofol shown to have accumulated in wild animals are small (Table 8). These amounts (<5 ppm wet weight) fall far below what could be perceived as dangerous based on much higher concentrations accumulated by test animals (Table 9). Ringed turtle doves accumulated DDE from the diet much more rapidly than dicofol (0.08% Cl-DDT as only DDTr in original dicofol product; Schwarzbach et al. 1988a), suggesting that dicofol is more readily metabolized. Dicofol is not extremely toxic to vertebrates. Nine acute and subacute toxicity tests (LC50 in water, dietary LC50, oral LD50) with eight species of fish, birds, and mammals showed that DDT was more toxic than dicofol products in seven instances, dicofol product was more toxic in one instance, and the chemicals were similarly toxic in one instance (Gaines 1960; Hill et al. 1975; Johnson and Finley 1980; Hudson et al. 1984). Furthermore, no deaths resulted when grackles were fed nominal 100 ppm (dry weight) Kelthane (0.4% DDE) for a month (Stickel and Reichel 1980), when domestic dogs were fed nominal 300 ppm (dry weight) Kelthane-NG for a year (Smith et al. 1959), or when rats were fed nominal 500 ppm (dry weight) Kelthane-NG for 2 years (Smith et al. 1959). Even though dicofol may cause slight-to-moderate thinning of eggshells, DDE seems to have greater negative effects on bird reproduction than dicofol. Finally, unlike DDT and its metabolite DDE, there is no evidence that any of dicofol's metabolites persist longer than dicofol itself, or that they have adverse wildlife effects.

There is still a factual basis for concern, however. Laboratory populations of grass shrimp experienced heavy mortality in water containing 14 ppb dicofol (Sharp et al. 1979), many amphipods died at 33 ppb (Spehar et al. 1982), and fathead minnows were similarly affected at 39 ppb (Spehar et al. 1982). Because dicofol was reported in San Francisco Bay at 30 ppb and the concentrations were believed to be higher in the Sacramento–San Joaquin Delta (Brocksen and Knight 1973), wild populations could also experience such mortalities.

The risk of biologically significant shell-thinning to sensitive species of fish-eating birds could be present at some locations. Fathead minnows accumulated 230 ppm (wet weight) dicofol from water containing 39 ppb and 81 ppm from water containing 19 ppb (Spehar et al. 1982); raptorial birds have shown slight-to-moderate eggshell thinning when fed 2.5 or 9.2 ppm (wet weight) dicofol (no detectable DDTr in original Kelthane; Wiemeyer et al. 1989; Clark et al. 1990). Species of concern include black-crowned night-herons (*Nycticorax nycticorax*), sandhill cranes (*Grus canadensis*), or even pelicans (*Pelecanus erythrorhynchos* or *P. occidentalis*) in southern Texas and the San Joaquin Valley of California.

An estimated nominal 84 ppm (dry weight) Kelthane-NG in the diet of female laboratory rats reduced their insemination rate, and an estimated nominal 110 ppm (dry weight) halved their pregnancy rate (Trifonova and Gladenko 1980); Kelthane-NG at a nominal 225 ppm (dry weight) prevented reproduction in laboratory rats (Brown 1972). Because dicofol residues in and on plant materials have measured as high as 94 ppm (probably wet weight; equivalent dry weight about 280 ppm; Stepanyan et al. 1984) or 400 ppm (probably wet weight; equivalent dry weight about 1,200 ppm; Nazer and Masoud 1986) immediately after spraying, and as high as 11 ppm (probably wet weight; equivalent dry weight about 33 ppm; Van Dyk 1975) 5 months after spraying, species of wild mammals could be at risk of reproductive impairment where dicofol is heavily used. Species of special concern, such as the endangered Fresno and giant kangaroo rats (*Dipodomys nitratoides exilis* and *D. ingens*), could be exposed in cotton agriculture in the San Joaquin Valley.

Chemical analyses of additional environmental samples taken where the chemical is heavily used could help to determine whether dicofol is harming wildlife. Samples should be collected in citrus groves in Florida and Texas, cotton fields in California and Arizona, and apple orchards in New York. Eggs of fish-eating birds would be useful sample material because field studies suggest that dicofol does get into fish, and evidence from the laboratory shows that high concentrations are possible. Concentrations in eggs associated with shell thinning are known from captive studies, which would make interpretation relatively easy. Also, if the species sampled were sensitive to chemical thinning of the eggshell, the eggshells themselves could provide direct evidence of effects. Eggs contain large amounts of fat that accumulates and concentrates organochlorine residues such as dicofol, making them easier to detect in chemical analysis. Presence of dicofol in eggs proves its presence both in the bird's diet and in its reproductive

organs. Finally, the historical example of DDE suggests that fish-eating birds should be studied. Eggs of raptors would probably be the next best material, and indeed the limited data gathered thus far show the highest concentrations in eggs of screech-owls (Table 8).

Once an understanding is gained as to what species at what sites receive the largest residue loads in their eggs, field studies can be undertaken to find significant associations between residue concentration and nest success, as was done with DDE (Blus 1984; Henny et al. 1984, 1985). Such studies, once they solve problems arising from the DDE that is present in eggs, should resolve most questions about dicofol's effects on birds.

Samples of nonflying small vertebrates such as fish, amphibians, reptiles, or small mammals would be useful and have the advantage of permanent association with the collection site. Fish are known to accumulate dicofol, and reptiles are promising because their eggs are heavily yolked. However, residue data from reptiles could, at present, be interpreted only by viewing them as diet for raptorial birds.

Finally, almost nothing is known about the effects of dicofol on nontarget members of agricultural invertebrate communities, and effects on nonagriculture invertebrate communities are unstudied.

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Appendix. Glossary

The following definitions reflect usage of terms for chemicals, toxicology, and pesticide residues in this analysis.

Aldrin 1,2,3,4,10,10-hexachloro-1,4,4*a*,5,8,8*a*-hexahydro-1,4:5,8-dimethanonaphthalene.

Carbaryl 1-naphthalenol methylcarbamate.

Carbofuran 2,3-dihydro-2,2-dimethyl-7-benzofuranol methylcarbamate.

Chlordane 1,2,4,5,6,7,8,8-octachloro-2,3,3*a*,4,7,7*a*-hexahydro-4,7-methano-1*H*-indene.

Chloro-DDT 1,1′-(2,2,2,1-tetrachloroethylidene)bis[4-chlorobenzene].

Cl-DDT chloro-DDT.

DCBH dichlorobenzhydrol, or 4,4′-dichlorobenzhydrol.

DCBP dichlorobenzophenone, or 4,4′-dichlorobenzophenone.

DCD dechlorodicofol; 4-chloro-*a*(4-chlorophenyl)-*a*(dichloromethyl)benzenemethanol.

DDD 1,1′-(2,2-dichloroethylidene)bis[4-chlorobenzene].

DDE (1,1′-dichloroethenylidene)bis[4-chlorobenzene].

DDT 1,1′-(2,2,2-trichloroethylidene)bis[4-chlorobenzene].

DDTr DDT, DDD, DDE, and Cl-DDT; r = residues.

Diazinon phosphorothioic acid *O,O*-diethyl *O*-[6-methyl-2-(1-methylethyl)-4-pyrimidinyl] ester.

Dicofol 4-chloro-*a*(4-chlorophenyl)-*a*(trichloromethyl)benzenemethanol.

Dicofol product A product containing dicofol as principal ingredient with dicofol metabolites such as DCBP, DCD, DCBH and DDTr also present; Kelthane is an example.

Dieldrin 3,4,5,6,9,9-hexachloro-1*a*,2,2*a*,3,6,6*a*,7,7*a*-octahydro-2,7:3,6-dimethanonaphth[2,3-*b*]oxirene.

Efficacy Effectiveness of a pesticide in controlling its target species.

Endrin 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4*a*,5,6,7,8,8*a*-octahydro-endo,endo-1,4:5,8-dimethanonaphthalene.

Heptachlor 1,4,5,6,8,8-heptachloro-3*a*,4,7,7*a*-tetrahydro-4,7-methanoindene.

HCB hexachlorobenzene.

Kelthane A commercial dicofol product consisting of dicofol plus lesser amounts of DDTr and dicofol metabolites such as DCBP, DCD, DCBH.

LC50 Concentration in ppm or ppb of active ingredient of toxicant that is lethal to 50% of the organisms exposed in a specified time interval. May be a concentration in water in which aquatic organisms are tested, or a concentration in the diet of birds or mammals.

LD50 Dose in milligrams of active ingredient of toxicant per kilogram of bird or mammal that is lethal to 50% of the animals exposed in a specified time interval.

Lindane (1*a*,2*a*,3*b*,4*a*,5*a*,6*b*)-1,2,3,4,5,6-hexachlorocyclohexane.

Malathion [(dimethoxyphosphinothioyl)thio]butanedioic acid diethyl ester.

Methoxychlor 1,1′-(2,2,2-trichloroethylidene)-bis[4-methoxybenzene].

Naled phosphoric acid 1,2-dibromo-2,2-dichloroethyl dimethyl ester.

ND Not detected in chemical analysis.

NG Designates a particular Kelthane or other dicofol product for which the percentage content of DDTr was not given in the publication referenced.

Nominal The concentration of a toxicant calculated to be present in a diet on the basis of amounts mixed. The actual concentration may differ when measured by chemical analysis.

Nulliparous Never having produced young.

ppb Parts per billion or µg/kg, ng/g, or µg/L.

ppm Parts per million or µg/g or mg/kg.

Propargite 2-(*p-tert*-butylphenoxy)cyclohexyl 2-propynyl sulfite.

Raptorial birds predatory falconiform and strigiform birds (eagles, ospreys, hawks, and owls).

Resistance a genetically conferred pesticide tolerance that may become common in a population of invertebrates after repeated exposure to the chemical.

Toxaphene chlorinated camphene.

Clark, Donald R., Jr. 1990. **Dicofol (Kelthane) as an Environmental Contaminant: A Review.** U.S. Fish Wildl. Serv., *Fish Wildl. Tech. Rep.* 29, 37 pp.

Dicofol is persistent in soil and on plants. No compelling evidence exists that dicofol breaks down or is metabolized to DDTr in nature. Dicofol does not accumulate in birds as rapidly as DDE, and it has reproductive effects that are less harmful than DDE. Fish, birds, and mammals are reproductively sensitive to dicofol products, but levels presently found in wildlife are below levels shown experimentally to cause significant harm. Eggs of fish-eating wild birds from citrus-, cotton-, and apple-growing areas should be analyzed for dicofol residues. Nest success of fish-eating birds in the most contaminated populations should be studied to evaluate the environmental effects of dicofol.

Key words: Contaminant, dicofol, Kelthane, toxicity, wildlife, reproduction, eggshell thinning.

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